The Journal of the American Medical Association

Published Under the Auspices of the Board of Trustees

VOL. 85, No. 26 CHICAGO, ILLINOIS DECEMBER 26, 1925

THE CLINICAL DIFFERENTIATION OF SPONTANEOUS INTRACEREBRAL HEMORRHAGE AND UREMIA

A FOURTH STUDY OF COMA*

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In 1915, one of us (E. R. L.) took part in a report [1] designed to help hospital interns and residents in recognizing the causes of coma so frequently dealt with by them as emergencies, especially in large urban hospitals. This was followed by two subsequent reports.[2]

These three reports concern 746 deaths, all with necropsies, and emphasize the infrequency of uremia as a cause of coma and the need for giving due value to the time under observation as an important factor in any discussion of errors in diagnosis. They do not, however, mention the importance of another factor, that death soon after entrance often prevents the examination and study of a puzzling coma by the more experienced attending staff, although in Holcomb's article,[2] this is suggested in a comment on the difference between the correctness of diagnoses made in the examining room and those subsequently made in the wards.

The present report has to do with a much smaller number, only thirty, and is limited to difficulties in distinguishing between spontaneous intracerebral hemorrhage and uremia, and to illustrating the value of the chemical examination of the blood for this purpose.

In the last four years (1921 to 1924, inclusive) at the Cook County Hospital, there have been thirty patients on whom chemical examinations of the blood were made, and death was found at necropsy to be due to spontaneous intracerebral hemorrhage. In nine of these (Group A), the clinical diagnosis was uremia, uremic coma or chronic nephritis; and in only three of these nine was cerebral hemorrhage or thrombosis mentioned as a possibility. For the remaining twenty-one (Group B), the diagnosis spontaneous intracerebral hemorrhage was correct in sixteen; and in five of these twenty-one, the incorrect diagnoses were skull fracture (two), poisoning, meningitis and diabetic coma. Thus, as shown in the accompanying chart, 53 per cent. of the diagnoses were correct, which may be compared with the 51 per cent. in 1915 and the 64 per cent. in 1921 in the reports mentioned.

The length of stay in the hospital for these thirty patients varied from an hour to twenty-two days, the average being about four and one-half days, so that there was usually sufficient time for the study of the illness. The relation between the time in the hospital and the accuracy of the diagnoses is graphically shown in the chart.

There were twenty-four men and six women, with ages from 24 to 77, averaging 51 in Group A, and 49 in Group B, which is significantly low. Nineteen patients were in coma on entrance, seven were semicomatose or stuporous, and four were conscious. In the first group, in which uremia was diagnosed, the average blood pressure was 205 systolic and 115 diastolic; the heart was enlarged in three; heart murmurs were heard in two, and definite palpable arteriosclerosis was noted in one. There was partial or complete hemiplegia in four, and in three no paralysis was found. The pupils were normal in two, and did not react to light in five. Urinalysis was done in eight, and albumin and casts were found in all of these; leukocytes were found in five, and Haines' solution was reduced in one. Wassermann tests of the blood or spinal fluid were made and found to be negative in eight; of the seven in which the spinal fluid was examined, blood was found grossly in six, and was entirely absent in one.

In the other group of twenty-one patients, the blood pressure varied from 125 to 208 systolic, and from 70 to 140 diastolic, an average of 194 and 109, respectively. In fifteen there were evidences of a hemiplegia; in three no paralysis was found. Heart murmurs were heard in two, and enlargement was noted in four. The pupils were normal in three, reacted to light in five, and did not react to light in thirteen. In seventeen urinalysis was done; albumin was found in fifteen, casts in nine and an increase of leukocytes in nine, and Haines' solution was reduced in six. The average specific gravity of the urine was 1.017. Spinal puncture of twenty patients revealed blood grossly in the spinal fluids of eighteen, a positive benzidin reaction in one, and an absence of any evidence of blood in the other. Wassermann tests were positive with two spinal fluids, but negative with the blood of one of these patients, and also negative with the blood of sixteen others so examined.

The alterations found by chemical examination of the blood [3] in Groups A and B are shown in Tables 1 and 2, respectively, and the averages of all cases in

* From the Cook County Hospital and the Pathological Laboratories of Rush Medical College.

1. Bissell, W. W., and LeCount, E. R.: A Consideration of the Various Forms of Coma, J. A. M. A. **64**: 1041 (March 27) 1915.
2. Bissell, W. W., and LeCount, E. R.: The Relative Frequency of the Various Causes of Coma, J. A. M. A. **68**: 500 (Feb. 17) 1917. Holcomb, Blair: The Causes and Diagnosis of the Various Forms of Coma, a Further Consideration, J. A. M. A. **77**: 2112 (Dec. 31) 1921.

3. This work was done by Dr. J. L. Kendall and assistants in the chemical laboratory at the Cook County Hospital.

Table 3. When the figures in these tables are compared with the averages for conditions in the blood in health and in disease (Table 4), it is evident that there is only an occasional patient in whom the increase in the substance sought in the blood at all approaches that found in uremia, and the averages for the whole series (with the exception of the blood sugar) are low in comparison with the estimates usually given for uremia. It is especially noteworthy that the averages with the illnesses wrongly diagnosed uremia are almost the same as the majority of those correctly diagnosed.

At the necropsies, all were found to have had intracerebral hemorrhages, and free blood was present beneath the leptomeninges in eight. Edema of the brain was noted in ten, and it may be of some significance as regards the symptoms that the illness in only four of these was correctly diagnosed. In two cases the kidneys differed in no way from the normal; a passive hyperemia was noted in nine, retention cysts in four, and cloudy swelling in five. In nine they were described simply as "chronic diffuse nephritis," in seven as small red granular kidneys, in two as primarily contracted, and in two as of the arteriosclerotic type. In twenty cases, or only 67 per cent., were obvious chronic pathologic processes present in the kidneys. The heart in five was normal grossly; in twenty-one there was myocardial hypertrophy; in eleven calcification, sclerosis or other changes in the heart valves, and in six a fibrous myocarditis. The arteries were sclerotic in varying degrees in twenty-three; fatty changes only were found in three, and syphilitic aortitis in four. In one the blood vessels were normal grossly.

In 1915, Bissell and LeCount [1] brought out the mistakes in the diagnosis of the causes of coma in their report of 200 patients who entered the Cook County Hospital, and whose bodies were examined post mortem. Of these, fifty-three died from spontaneous intracerebral hemorrhage, of which twenty-seven cases, or only fifty-one per cent., were correctly diagnosed. Forty-six of the 200 cases were diagnosed clinically as uremia, and in sixteen of these, or 35 per cent., death was the result of cerebral hemorrhage, and in only eight, or seventeen per cent., of uremia. In a similar article [2] in 1917, the same authors published reports of 200 additional patients who entered in coma and died; of the entire 400, death in ninety-five was due to cerebral hemorrhage, and in nineteen to uremia, indicating that in this class of patients the relative frequency of death due to cerebral hemorrhage to that from uremia is about 5 to 1.

In the diagnosis of spontaneous cerebral hemorrhage many so-called "focal" symptoms have been observed, as, for instance, those emphasized by Cumston,[4] dilatation of the pupil, papilledema, ptosis of the upper lid, corneal anesthesia and conjunctival hyperemia, all on the side of the lesion; but these are also often found on the side opposite the hemorrhage, and some of them may be observed in uremia, so that their value is not great. The blood pressure is usually high in both conditions except in the terminal stages. Shaw,[5] in a study of essential hypertension (hyperpiesia), came to the conclusion that all the manifestations of this condition are also encountered in uremia, and that the symptoms of hyperpiesia which simulate uremia are produced by toxins present in the blood as the result of some extrarenal inflammation, rather than to metabolites retained because of disorders of the kidneys. This author and O'Hare [6] have each described a case in which there were clinically high blood pressure and myocarditis (?), and the necropsies revealed spontaneous intracerebral hemorrhage with no renal disease. Williams [7] concludes that there is no relation between the blood pressure and the nonprotein nitrogen of the blood, and that arterial hypertension with normal nonprotein nitrogen blood values and normal kidney excretion does not justify the clinical diagnosis of chronic nephritis. This is of significance because with coma of obscure origin, when there is evidence suggesting a chronic nephritis, it certainly is true that altogether too frequently the results

TABLE 1.—*Chemical Analysis (Milligrams per Hundred Cubic Centimeters of Blood) of the Blood for Nine Patients Dying from Spontaneous Intracerebral Hemorrhage with the Clinical Diagnosis of Uremia*

	Nonprotein Nitrogen	Urea Nitrogen	Uric Acid	Creatinin	Sugar
Range	34.56 to 49.97	13.07 to 28.02	2.76 to 4.00	1.45 to 1.51	104 to 244
Average	38.84	18.35	3.33	1.49	178

TABLE 2.—*Chemical Analysis (Milligrams per Hundred Cubic Centimeters of Blood) of the Blood of Twenty-One Patients* Dying from Spontaneous Intracerebral Hemorrhage*

	Nonprotein Nitrogen	Urea Nitrogen	Uric Acid	Creatinin	Sugar
Range	28.0 to 64.2	7.93 to 46.7	1.58 to 3.16	1.40 to 2.50	101 to 333
Average	36.74	19.85	2.38	1.65	164
Number of cases	13	21	15	20	12

*Sixteen cases were diagnosed correctly.

TABLE 3.—*Conditions in the Blood of Thirty Patients (Milligrams per Hundred Cubic Centimeters)*

	Nonprotein Nitrogen	Urea Nitrogen	Uric Acid	Creatinin	Sugar
Range	28.0 to 64.2	7.93 to 46.7	1.58 to 4.00	1.40 to 2.50	101 to 333
Average	37.79	19.10	2.86	1.57	171
Number of cases	18	27	20	26	17
Percentage of increase above normal*	20%	19%	43%	10%	71%
Percentage of increase above normal in uremia*	739%	1,150%	775%	1,370%	50%

* Normal being the averages of the amounts of the various chemical elements of the blood in health as given in a previous reference.

TABLE 4.—*Averages of the Chemical Examination of the Blood in Health and in Uremia* *

	Mg. per 100 C.c. of Blood				
	Nonprotein Nitrogen	Urea Nitrogen	Uric Acid	Creatinin	Sugar
Normal	25 to 35	12 to 30	1 to 3	1 to 2	100
Uremia	90 to 250	70 to 300	4 to 27	4 to 33	100 to 200

* From Myers, V. C.: Practical Chemical Analysis of Blood, St. Louis, C. V. Mosby Company, 1924, p. 16. Hawk, P. B.: Practical Physiological Chemistry, Ed. 8, Philadelphia, P. Blakiston's Son & Co., 1923, p. 273.

4. Cumston, C. G.: Intracranial Hemorrhage, Internat. Clin. **3**: 89, 1917.

5. Shaw, H. B.: High Blood Pressure and Relationship to Uremia and Hyperpiesia, Lancet **2**: 1307 (Dec. 24) 1921.

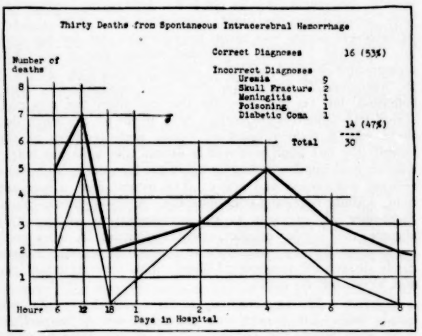
6. O'Hare, J. P.: Case of Vascular Hypertension with Cerebral Hemorrhage and Angina Pectoris, M. Clin. N. Am. **4**: 1767 (May) 1921.

7. Williams, J. L.: Total Nonprotein Nitrogen Constituents of Blood in Arterial Hypertension, Arch. Int. Med. **27**: 748 (June) 1921.

of the chemical examination of the blood are disregarded, and the coma is considered uremia.

The presence of albumin and casts in the urine is usually and correctly interpreted as evidence of renal disease, or, as Senator [8] has said, "the more closely the kidneys are examined in cases of albuminuria, the oftener are structural changes found," but "it has been noted by a large number of observers that in various pathologic conditions of the nervous system, such as epilepsy, delirium tremens, various psychoses, cerebral apoplexy, nervous exhaustion, migraine, and Basedow's disease, transient albuminuria occurs with or without hyaline casts and sometimes with casts alone, and this in cases where there is no chronic renal disease or passive congestion of the kidneys such as may occur in convulsions." The transient albuminuria occasionally occurs in healthy persons following simply excitement.

Blood is usually found in the spinal fluid with intracranial hemorrhage, but may not be present when the lesion is deeply located. However, if the fluid is examined about three days after the "stroke," it usually has a canary-yellow color which often persists for months, although the ordinary tests for blood remain negative.[9]



Correct and incorrect diagnoses in thirty deaths from spontaneous intracerebral hemorrhage: Heavy line, deaths; light line, number correctly diagnosed. Three deaths not plotted occurred: one on the twelfth, and two on the twenty-second day; two of these were correctly diagnosed.

Volhard and Fahr [10] believe that the name uremia should be applied only when symptoms are observed of true urinary poisoning with definite evidence of serious impairment of the kidneys, the diagnosis to be made from the symptoms and results of laboratory studies. According to McElroy,[10] such laboratory examinations demonstrate a "marked retention of the residual blood nitrogen and urea, a low specific gravity of the night urine, a low fixed specific gravity of the day urine, low salt and nitrogen concentration, a low phenolsulphonephthalein excretion, a low urea index, a high Ambard coefficient, and increased osmotic tension [11] of the blood. But in addition to these more or less typical pictures, there is a smaller number of cases in which functional kidney tests give very variable results in the different manifestations of uremia. In the convulsive attacks associated with the acute and second stage of diffuse glomerulonephritis, and . . . with the hypertension of sclerotic conditions, there is usually very little evidence of kidney insufficiency as indicated by the tests now generally used. In such cases, the nonprotein nitrogen and urea in the blood are within normal limits." These two constituents of the blood are usually in about the relation of 2 to 1; that is, the urea nitrogen constitutes about 50 per cent. of all the nonprotein nitrogen of the blood in health, whereas with retention such as is observed in the so-called chronic interstitial nephritis, the urea nitrogen may be as much as 75 or 80 per cent.

8. Senator, H.: Diseases of the Kidney, in Nothnagel's System of Medicine, Philadelphia, W. B. Saunders Company, 1905, p. 41.

9. Harris, W.: Diagnostic Value of Lumbar Puncture in Cerebral and Spinal Hemorrhage, Brit. M. J. **1**: 635 (April 22) 1922.

10. Volhard and Fahr, cited by McElroy: in Diseases of the Kidneys, Tice's System of Medicine, Hagerstown, Md., W. F. Prior Company, Inc. **6**: 570, 1925.

11. This is contradicted by Schade and Claussen (Der onkotische Druck des Blutplasmas und die Enstehung der renal bedingten Odeme, Ztschr. f. klin. Med. **100**: 363, 1924), who have found that lessened osmotic pressure of the blood due to alterations in the protein content ("hypoonkie") is a specific symptom of nephritis with edema.

CONCLUSIONS

The following conclusions pertain simply to the condition observed in this series of thirty deaths from intracerebral hemorrhage:

1. No patient dying from intracerebral hemorrhage with the clinical diagnosis of uremia had a nitrogen retention comparable to that of true uremia.

2. In spontaneous intracerebral hemorrhage, the increase above the normal of the various nitrogenous elements of the blood varied from 16 to 43, the creatinin (16 per cent.) being the least increased, and the uric acid (43 per cent.) the most.

3. In 67 per cent. of the thirty, the gross changes in the kidneys indicated some chronic disease; in 7 per cent., no gross changes of any kind were found.

4. The presence of blood in the spinal fluid and the great increase in the nitrogenous elements in the blood are apparently the two most important factors in the differential diagnosis between uremia and spontaneous intracerebral hemorrhage.

TREATMENT OF SEPSIS WITH MERCUROCHROME-220 SOLUBLE

REPORT OF NECROPSIES IN FIVE CASES *

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In the last few years a new antiseptic, mercurochrome-220 soluble, has been placed on the market, and considerable literature extolling its virtues has accumulated. Only two or three meager articles have come to my attention reporting unfavorable symptoms following the intravenous administration of this preparation and advising more caution and control in its application.

During the past year the following five cases, in which mercurochrome was administered intravenously during life, came to necropsy in the chief medical examiner's department of the city of New York, and showed, among other anatomicopathologic changes, the lesions of mercurial poisoning:

CASE 1.—*History.*—Mrs. A. S., aged 36, white, was admitted to the hospital, March 31, 1924, with a history of having had the uterus opened to induce abortion, and of taking a large amount of various abortifacients. The chief symptoms, on admission, were pain in the lower part of the abdomen, vaginal bleeding, nausea and fever; the temperature on admission was 102 F.; the pulse, 96. Blood examination revealed white blood cells, 14,800; polymorphonuclears, 87 per cent.

* Read at the Thirty-Seventh Annual Meeting of the American Association of Genito-Urinary Surgeons, Washington, D. C., May 4, 1925.

* From the pathologic laboratories of Bellevue Hospital, Dr. Douglas Symmers, director, and the Office of the Chief Medical Examiner of the City of New York, Dr. Charles Norris, chief medical examiner.

April 3, the blood culture was positive for *Streptococcus hemolyticus* (2 colonies per cubic centimeter). Gentian violet was administered without beneficial effect. Blood cultures were positive for *Streptococcus hemolyticus*, April 13, 19, 24 and 28, there being, respectively, 6, 10, 14 and 20 colonies per cubic centimeter. April 25, 35 c.c. of a 1 per cent. solution of mercurochrome was administered; similar quantities were given, May 9 and May 11. May 12, 20 c.c. was administered, and again, May 14, 35 c.c.. No change was apparent following the administration of mercurochrome, either in temperature or in the course of the illness, save for a discoloration of the skin. The urine at various times contained albumin, pus and red blood cells. Death occurred May 15.

Necropsy.—The peritoneal cavity contained about 50 c.c. of dark straw-colored fluid. The left pleural cavity was about one third filled with free, bloody, serofibrinous fluid. The kidneys were large and swollen; on section, the cortex was very pale and the medulla deeply injected; the capsule was free, the surface smooth and pale yellowish; the pelves, ureters and urinary bladder were normal. There were extensive adhesions about the loops of the small intestine opposite the lower right abdominal quadrant, where it was firmly attached to the wall. The mucous membrane of the small intestine was natural in appearance, the upper portion being filled with yellowish bile-stained fluid. The cecum and ascending colon were enormously distended with gas; the mucosa showed elevated reddish rugae and, in places, a distinct granular appearance. The mucous membrane of the transverse and descending portions showed grayish and granular areas with superficial ulcerations. The same was true of the lower portion of the rectum, namely, that portion above the anal canal (suggestive of mercuric chlorid). The other organs showed nothing worthy of note.

The anatomic diagnosis was: serohemorrhagic pleuritis (left); collapse of the left lung; splenomegaly; infarction of the spleen; necrosis of the spleen; ulcerative and exudative colitis; acute parenchymatous nephritis; parenchymatous degeneration of the viscera; sepsis.

On histologic examination, the kidneys showed degeneration of the renal parenchyma, with hemorrhage, fatty infiltration and calcium deposits in the descending tubules. The ulcers in the intestine showed necrosis of the surface epithelium, with round-cell infiltration at the margins and diffuse edema and hemorrhagic extravasation.

CASE 2.—*History.*—Mrs. M. A., aged 26, colored, who was admitted to the hospital, June 7, 1924, had had an abortion performed three weeks prior to admission. The patient developed pain over the lower part of the abdomen, chills and fever. On admission, her temperature was 102 F.; pulse, 104; respiration, 24; the blood culture was sterile; albumin and a few pus cells were found in the urine. June 11, 20 c.c. of a 1 per cent. solution of mercurochrome was given intravenously, after which the patient developed severe diarrhea, lasting two days, and albumin, pus, casts and blood cells were present in the urine. June 17, 20 c.c. of a 1 per cent. solution of mercurochrome was administered intravenously; the patient at once developed severe chills, and shortly thereafter diarrhea, which persisted until death, June 18. The blood culture at this time was sterile.

Necropsy.—The pelvic cavity showed a small amount of purulent material with a few flakes of fibrin, and the lower portion of the ileum showed discolorations and soft areas covered by fresh fibrin. The abdominal cavity contained a small amount of fluid, which was slightly cloudy and tinged yellow. The liver weighed 2,080 gm.; a cut section showed small yellowish mottled areas, suggestive of focal necroses. The gallbladder contained dark greenish, ropy bile, and the mucous membrane was normal. The kidney capsule was stripped with some difficulty. The surface was studded with small hemorrhages. The kidney substance was mottled; the markings were very irregular and the pyramids slightly violaceous. The pelves and ureters showed no changes. The combined weight of the kidneys was 360 gm. The uterus measured 3½ by 2⅝ by 1⅝ inches. On section, the mucous membrane was covered with a blood clot and was injected; the sinuses were somewhat enlarged, but there was no evident perforation. The stomach was normal. The small intestine was normal as far down as the last half of the ileum, where there were injected areas. The last foot of the ileum showed distinct ulcers, covered with greenish slough, and the wall of the ileum was thickened. The upper portion of the large intestine and the lower portion of the sigmoid showed petechial hemorrhages. The rectum presented ulcers similar to those found in the ileum; namely, irregular, serpiginous ulcers, covered by greenish slough. The other organs showed nothing of interest in the present connection.

The anatomic diagnosis was: septic pelvic peritonitis secondary to abortion; ulcerative enteritis and colitis; cardiac hypertrophy; bronchopneumonia; acute diffuse nephritis; sepsis.

Histologic examination of the kidney showed intense parenchymatous degeneration, with necrosis, round-cell infiltration, hemorrhage and edema. The intestine showed necrosis and degeneration of the surface epithelium, and replacement by edematous and necrotic slough. The deeper portions of the intestinal wall were edematous and showed dense collections of nuclear débris and, in places, hemorrhagic extravasation.

CASE 3.—*History.*—M. M., a white woman, was admitted to the hospital, Sept. 24, 1924, with a diagnosis of septic abortion (spontaneous) ten days before admission, followed by chills, fever and abdominal pain. September 25, 20 c.c. of a 1 per cent. solution of mercurochrome was given intravenously at 8 p. m., followed by chill and a rise of temperature to 104.6 F., with a subsequent fall to 101.4 F.; four very loose stools were passed during the night. September 26, at 8 p. m., 1 c.c. of typhosus serum was given intravenously for foreign protein, followed by chill; an alcohol sponge was given. The temperature dropped to 99.6 F. within a few hours. Previous to September 27, the patient had been irrational and semicomatose, but on that day, for the first time, she was conscious and talked intelligently. At 9 p. m. of the same day there was a sudden onset of coma, with frothing at the mouth; the left pupil was widely dilated, the right not markedly so, both pupils reacting to light; the knee jerks and elbow reflexes were equal and active. The patient died within one hour, without regaining consciousness. A blood culture taken, September 25, previous to the administration of mercurochrome, was sterile; another, taken, September 26, after mercurochrome therapy, was likewise sterile. September 26, the stools showed the presence of blood. The urine contained pus and blood, but no casts.

Necropsy.—The pericardial cavity contained yellowish, cloudy fluid. The parietal and visceral layers of the pericardium were rough and covered with a shaggy exudate. The kidneys were large, weighing 410 gm.; the capsule was stripped with some difficulty; the cortex was wide and pale; the pyramids, violaceous; the pelves and ureters were stained yellowish but were otherwise normal. Watery, blood-stained fluid was present in the large intestine, the mucous membrane being markedly injected in some places, other portions showing raised, greenish gray ulcerations and marked thickening of the bowel and the whole being stained reddish. The scalp, calvarium and meninges were normal. The left hemisphere was much softer than the right, and the markings between the gray and white matter were not as sharp as on the right side. Careful search revealed a formed embolus in the middle cerebral artery on the left side, just above the bifurcation of the carotid and anterior to the middle third. The base of the skull was normal. The other organs showed nothing of interest in the present connection.

The anatomic diagnosis was: embolus, left middle cerebral artery; acute septic valvulitis, involving the aortic valve; anemic infarct of the heart muscle; anemic infarct of the left kidney; septic spleen; parenchymatous degeneration of the heart muscle, liver and kidney; ulcerative colitis; septic endometritis; corpus luteum, left ovary; acute pericarditis; pyopericardium; mercurochrome staining of the viscera; sepsis.

On histologic examination, the kidneys showed a diffuse parenchymatous degeneration, with fat deposition and extensive calcium degeneration of the descending tubules. The colon showed superficial ulcerations with epithelial necroses and an edematous slough. The remainder of the wall showed intense edema, hemorrhagic extravasations and granular débris.

CASE 4.—*History.*—T. P., a white man, was admitted to the hospital, Nov. 1, 1924, in delirium, with an infected right hand, spasticities, a temperature of 103.2 F., pulse 96, and respirations 26. The urine contained albumin and a few hyaline casts, but no pus or blood cells. The hand infection was treated by incisions and drainage; a culture of the wound showed the presence of streptococci. November 4, blood culture was positive for streptococcus, and on the same day 20 c.c. of a 1 per cent. solution of mercurochrome was administered intravenously, followed in one and one-half hours by a severe chill and an elevation of temperature to 105 F. On the following morning, the temperature receded to 101.2 F. and severe diarrhea developed, which persisted until death, November 19. The patient's general condition showed no improvement. He presented quite marked puffiness of the eyelids and the urine gave a marked reaction for albumin, many hyaline and granular casts, and numerous pus and blood cells. November 15, 20 c.c. of a 1 per cent. solution of mercurochrome was given intravenously, the reaction on this occasion being less marked than on November 9. November 17, the blood culture was positive for streptococci (from 20 to 30 colonies per cubic centimeter of blood).

Necropsy.—Externally there were no injuries, except for two operative incisions on the dorsum of the right hand. The deep tissues were necrosed, and the muscle was deep red and infiltrated with pus; no thrombosis was noted in the surrounding vessels. The right pleural cavity contained purulent fluid, and the right lung was covered with fresh fibrin over the base, and was injected; the cut surface was congested. The left lung was free and the cut surface normal, except for three or four small abscesses on the surface. The pericardial sac was distended by purulent fluid, and both the parietal and visceral layers of the pericardium showed a shaggy membrane. The valves were normal; the musculature was normal, except for a small abscess cavity in the fat of the apex, not involving the muscle; the fossa ovalis was closed. The kidneys were large and swollen; the markings were somewhat obscured. The cortex was pale yellowish pink, and the pyramids were deep purplish red; the pelves were injected. The esophagus was normal. The stomach contained yellowish fluid. The mucous membrane was normal. The small intestine contained the usual chyme; the mucous membrane was normal. The large intestine contained fluid greenish material; the mucous membrane from the ileocecal valve through to the rectum showed superficial ulceration and necrosis, resembling mercuric enteritis. The brain showed a purulent exudate covering both hemispheres, most marked on the left side and extending into the medulla and spinal cord; smear of the pus showed many streptococci. The other organs showed nothing of interest in the present connection.

The anatomic diagnosis was: acute suppurative pericarditis; acute suppurative pleurisy (right side); acute suppurative meningitis; abscess of the lung and pericardium; cellulitis of the right hand; acute ulcerative colitis; acute nephritis.

On histologic examination, the kidney showed intense parenchymatous degeneration, marked injection of the vessels, fatty infiltration of the cells and some calcium degeneration. The colon showed erosions of the epithelium, localized ulceration and edema, and degeneration of the submucosa and muscularis, with extensive nuclear débris.

CASE 5.—B. G., a white woman, aged 36, was admitted to the hospital, Nov. 17, 1924, with the diagnosis of septic abortion, resulting from the self-introduction of an orange-wood stick six days prior to admission. During her stay in the hospital, the temperature ranged from 104 to 105 F., the pulse from 105 to 130, and the respirations from 26 to 48. The white blood cells numbered 15,600; polymorphonuclears, 88 per cent. The urine contained albumin, an occasional cast and pus cells. November 18, the blood culture was positive for *Streptococcus hemolyticus* and *Staphylococcus aureus.* November 19, 20 c.c. of a 0.5 per cent. solution of mercurochrome was given, and on the following day a similar amount was administered. Blood culture taken after the latter injection was still positive, and the urine contained albumin, pus, many casts and red blood cells.

The patient died, Novemebr 21.

Necropsy.—Puncture wounds were noted in the left and right cubical fossae, under the breasts. The vaginal orifice was wide, admitting three fingers with ease; a brownish discharge escaped from it. Milk could be expressed from the breasts. On section, no free fluid was present in the peritoneal cavity; the diaphragm reached the fourth rib on both sides. The heart weighed about 400 gm.; the pericardium showed a number of petechial hemorrhages. The mitral valve was thickened from an old mitral valvulitis and, in addition, showed fresh vegetations; the rest of the valves were normal; the fossa ovalis was closed. The spleen was large, weighing 300 gm.; on section it showed a large infarct, with softening in the center of the infarct. The kidneys were large, weighing 428 gm.; the capsule was stripped with some difficulty. The surface was studded with small abscesses, surrounded by hemorrhagic zones. On section, the cortex was pale yellowish, with deeply injected medulla; the pelves and ureters were normal. The uterus measured 4 by 3 by 2½ inches; there were two grayish punctate marks on the anterior lips of the cervix, probably tenaculum marks. The tissue of the internal os was greenish and necrotic. In the cavity of the uterus was a placental site on the superior wall, covered by a fresh, green, necrotic slough. The walls of the uterus were thick and the sinuses dilated. The left ovary showed a large corpus luteum cyst, measuring 1 inch in diameter, filled with clear fluid and surrounded by a lutein zone, 1/8 inch in diameter. The stomach contained brownish fluid; its mucous membrane was normal. The small intestine contained the usual chyme and feces; the mucous membrane was normal throughout, except for punctate hemorrhages here and there. The cecum was intensely injected, hemorrhagic and stained yellowish red; the lower colon showed a half dozen superficial ulcerations. The other organs showed nothing of interest in the present connection.

The anatomic diagnosis was: acute and chronic mitral valvulitis; infarcts of the spleen; septic endometritis; acute colitis; acute nephritis; miliary abscesses of the kidneys.

Histologic examination revealed a slough over the erosions in the colon, edema, some polynuclear and round-cell infiltration, and hemorrhage. The kidneys showed parenchymatous and fatty degeneration, focal abscesses and marked congestion.

COMMENT

These five cases represent the first of a series that came under observation in the chief medical examiner's office. Since these cases all showed strikingly similar lesions, both in the kidneys and in the colon, the chief medical examiner has performed postmortem examinations on all cases reported to his department in which mercurochrome has been used, with the result that he has accumulated a series of twelve cases, all of which showed evidence of renal and colon involvement.

In the five cases here presented, it is our opinion that mercurochrome induced the intense nephritic and intestinal lesions and thus contributed to death. Chemical analysis of all the viscera in these cases, made by A. O. Gettler,[1] pathologic chemist to Bellevue Hospital and toxicologist to the chief medical examiner of New York, showed mercury in even larger amounts than were found in mercuric chlorid poisonings, a fact indicating the marked toxicity of this drug and justifying the opening statement.

It may be argued that cases have been reported in which the drug was used successfully, although a temporary nephritis and colitis did accompany its use, and hence the lesions could not be of moment. To this, it may be answered that in cases of mercuric chlorid poisoning with nonsecreting kidneys and severe colitis patients do recover and the kidney and the colon are apparently restored to normal, though subsequently these cases show a chronic nephritis, a factor that has not as yet been determined in mercurochrome cases.

1. Gettler, A. O.: To be published.

From the meager cautioning literature, and the unfavorable cases that are everywhere discussed but not published, it is evident that the dangers of the drug have not been brought sufficiently to the fore. Locally, mercurochrome probably is a very good antiseptic; systemically, it may possibly be of value in selected cases when properly administered and controlled, though its indiscriminate use, for example, in such conditions as acute rheumatic fever or malaria, should not be countenanced. That its dangers are constanly to be borne in mind is apparent from the foregoing case reports, in that mercurochrome in its therapeutic dose not only exhibited no curative effect, but produced lesions associated with a toxicologic agent, and the margin of safety between a therapeutic and a toxic dose must be variable and small indeed.

MANGANESE POISONING AND ITS EFFECT ON THE CENTRAL NERVOUS SYSTEM

REPORT OF SIX CASES *

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My attention was recently attracted to the subject of managanese poisoning when I was consulted by a patient who was suffering with a malady, the clinical picture of which was not at all familiar to me. The condition was obviously the result of a disease of the central nervous system, but it fitted no classic picture of a well recognized syndrome. In view of the fact that the patient had worked for several months in a manganese grinding plant, the possibility of manganese poisoning was considered. The descriptions available of this disease agreed so well with the subjective and objective symptoms of the patient under consideration that this diagnosis was made, but with some hesitation. Later, the wife of the patient volunteered the information that she knew of several other workers in the same manganese grinding plant who suffered similarly. Forthwith, a trip was made to the neighborhood of this grinding plant in Virginia, and five similar cases were found.

HISTORY

In looking up the subject, one is surprised that there are so few cases of manganese poisoning recorded and that so little has been written about the disease. Couper,[1] in 1837, first described the disease among workmen who handled manganese dioxid in the manufacture of chlorin for bleaching powder. The description of the symptoms of his patients was very similar to those in my series. In 1901, von Jaksch [2] described three cases that he considered atypical types of disseminated sclerosis in workmen employed in manganese grinding plants. He attributed the disease to a manganese toxicosis. Embden,[3] Friedel,[4] Seiffer [5] and Seelert [6] noted the disease and described cases similar to those already reported. Von Jaksch,[7] in 1907 and 1910, reported other cases. In all, only fifteen cases have been reported to date in the European literature.

In 1913, Casamajor [8] reported nine cases, the first ones to be recorded in America. In 1919, Edsall, Wilbur and Drinker [9] completely summarized the literature and reported in detail several of their cases. In 1922, Davis and Huey [10] added two cases to those already recorded.

With the exceptions of Couper's five cases and four of von Jaksch's, all of the cases reported have been caused by inhalation, or swallowing in the saliva, of manganese dust in grinding plants. Von Jaksch [7] states that no new cases developed when the dust was abolished, and it is his belief that the lung is the portal of entry, the manganese later entering the nervous system probably as an albuminate.

My six patients had all been in contact with an atmosphere heavily laden with fine manganese dust for periods varying from three to ten months. Not until recently had any precaution been taken to protect the men working in this mill. After several cases of suspected poisoning had developed, the operators of the plant had provided the workmen with masks, but as they were uncomfortable, the masks were seldom worn. Dust collectors were provided in the mill, but they were entirely inadequate to collect the dust produced in the process of grinding the ore.

PATHOLOGY

Little is known of the exact pathology of the disease. There were no postmortem examinations in any of the foreign cases; in fact, there is no report in the literature of the disease, per se, ever having been fatal. Casamajor [8] reported a necropsy on one patient with manganese poisoning who died of pneumonia. He noted no gross changes in the brain or cord but stated that he found the longitudinal fibers of the pons degenerated. Unfortunately, no mention was made in the report of this necropsy of the microscopic appearance of the corpus striatum. It is my belief, based on clinical findings and the experimental work of Mella,[11] that the central nervous system lesion of manganese poisoning is probably in this location. The similarity of the poisoning to Parkinson's disease and to Wilson's syndrome is striking. Stocker [12] states that in making the diagnosis of Wilson's syndrome (progressive lenticular degeneration) one must be careful that the patient has not worked in manganese, as the poisoning from this element strikingly resembles such a condition.

Mella,[11] in an experimental study of poisoning by manganese in monkeys, found a clinical picture that he attributed to lesions of the basal nuclei. Histologic examination in his cases showed definite changes, particularly in the corpus striatum and globus pallidus.

SYMPTOMS

The clinical picture of chronic manganese poisoning is such a striking one that, when once seen, it is hard to confuse with other diseases of the central nervous system. The initial symptom in three of my six patients was disturbance of gait and in two of the remaining

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1. Couper: J. de clin. méd., de pharm. et de toxicol. **3**: 223, 1837.
2. Von Jaksch, R.: Wien. klin. Rundschau 1901, p. 729.
3. Embden, H.: Deutsch. med. Wchnschr. **27**: 795, 1901.
4. Friedel: Ztschr. f. Med. Beamte **15**: 614, 1903.
5. Seiffer: Berl. klin. Wchnschr. **41**: 370, 1904.
6. Seelert, H.: Monatschr. f. Psychiat. u. Neurol. **34**: 82, 1913.
7. Von Jaksch, R.: München. med. Wchnschr. **54**: 969, 1907; Die Vergiftungen, 1910, Vienna and Leipzig, p. 231.
8. Casamajor, Louis: An Unusual Form of Mineral Poisoning Affecting the Nervous System: Manganese? J. A. M. A. **60**: 646 (March 1) 1913.
9. Edsall, D. L.; Wilbur, F. P., and Drinker, C. K.: J. Indust. Hyg. **1**: 183 (Aug.) 1919.
10. Davis, G. G., and Huey, W. B.: J. Indust. Hyg. **3**: 231 (Dec.) 1922.
11. Mella, Hugo: Experimental Production of Basal Ganglion Symptomatology in Macacus Rhesus, Arch. Neurol. & Psychiat. **11**: 405 (April) 1924.
12. Stocker, W.: Ztschr. f. d. ges. Neurol. u. Psychiat. **15**: 251, 1913.

patients this was the second manifestation. In each of the patients the following symptoms were invariably found: nervousness; weakness and fatigue; disturbed gait with retropulsion on arising and propulsion on walking, and causeless laughter with silly conduct and expression. Paresthesias were noted as an early complaint in four cases; intention tremor of the hands, in five; monotonous speech, in five; awkward, clumsy movements of the extremities with loss of the finer movements of the hands, in four; masked expression, in four; a loose-hanging lower jaw, in three; an infrequent, deep inspiratory sigh, in three, and lethargy, in two. Abnormal mental symptoms were observed in every patient, mostly of personality, and in only one case were intellectual aberrations observed. Irritability, lack of sociability, tearfulness and mild exaltation were the outstanding changes. Two of the patients became suspicious, without cause, of their families and friends. The organic neurologic findings were almost uniform, the gait was altered in every case, the station was unsteady and the deep tendon reflexes were increased over the normal in the majority. Intention tremor of the hands and increased muscle tone were found together with masked expression and monotonous speech. None of the patients showed pathologic plantar reflexes, objective sensory changes, atrophy or edema. The pupils were not altered in size, shape or reaction. The fields of vision were normal as well as could be detected by a rough examination. Intra-ocular examination showed definite pallor in one case and a very reddened vascular retina in another. No involvement of the cranial nerves was noted except suspicious weakness of the facial nerve in one patient and the drooping lower jaw, in three.

Peripheral neuritis is a frequent symptom of poisoning from various metals, and it is possible that there may be some neuritic involvement in these cases. This may be suspected in view of the fact that four of the patients examined complained of paresthesias of the extremities, and in two of them actual pain was described. Against this belief is the activity of the tendon reflexes, the absence of objective sensory changes and muscular atrophy, and the character of the pain.

An interesting feature of the investigation of these patients was that the operators of the grinding plant and a few people in the community were of the opinion that the symptoms were due to the drinking of corn whisky and fermenting cider. Adjacent to the manganese mill was a vinegar plant, and it was stated that the mill workers would steal the fermenting cider and become intoxicated by it. One of the patients reported here had been sent to one of the state insane hospitals as an alcoholic, and his condition while there also was thought to be due to the use of alcohol. It is a fact that, with one exception, each of these patients drank rather heavily, at one time or another, while in contact with the manganese dust. In studying the case reports, however, it will be noted that Patient 5 developed symptoms of poisoning after being in contact with manganese dust for a shorter period of time than the others; but from his own statements, from the statements of his physician and from other reliable sources, the information was gained that he had never used alcohol in any form as a beverage. There is nothing to convince me that alcohol played any part in the symptom complex here presented. Certainly, I have never observed or seen reported, either in acute or in chronic alcoholism, a description that would fit the picture of these cases.

REPORT OF CASES

CASE 1.—*History.*—A white man, aged 35, who had been a farmer prior to working in a manganese grinding plant, where he was employed from March until December, 1924, was in constant contact with the manganese dust and not until September did he wear a mask for protection. His family history was negative except that one brother had symptoms similar to those of which the patient complained, and he also had worked in the same manganese plant.

The past history was negative except for a slight attack of influenza in 1918, and there was no record of venereal disease. The patient's habits were good except that he had not slept well since working in manganese and during this time he drank corn whisky rather heavily. In July, 1924, he first noticed that he was nervous, unable to sleep, and had a sense of falling forward when walking, and on arising had a tendency to stagger backward. There were paresthesias in the arms and legs, and aching pain in the right arm and leg. He developed a slow, monotonous speech, and there was some loss of the fine movements of the hands, more noticeable on the right. The patient developed tremor of the hands, which was more perceptible on movements of intention. His memory was impaired; he became mentally confused at times, was silly, was constantly smiling and would laugh without cause. Occasionally there was vertigo. The general physical examination was entirely negative except that he showed evidence of pyorrhea alveolaris.

Neurologic Examination.—The patient had a slight sway in station, showed a tendency toward propulsion in walking and noticed that the movement of the right leg was awkward. On arising, he would tend to fall backward. The deep reflexes were exaggerated, and there were no pyramidal tract signs. There was a fine tremor of the extended fingers and definite intention tremor of both hands; sensation was normal. Speech was slow and monotonous, and there was a masked, stolid expression. The pupils were equal, regular and reacted promptly; there was no nystagmus. The cranial nerves were intact. The fundi showed no changes, and the vision fields were not restricted, as tested by rough examination.

At the time the patient left the manganese plant, his wife stated that he showed definite confusion. He was asocial, listless, could not sleep, dreamed a great deal and frequently would scream out in his sleep. He was emotionally disturbed and this was manifested by tearfulness, irritability and a constant, causeless laughter. At the time of examination, he showed no mental symptoms other than silliness, tearfulness, lack of sociability and the causeless laughter.

CASE 2.—*History.*—A youth, aged 18, whose family history and past history were relatively unimportant, had worked in a manganese grinding plant where he was in constant contact with the dust for four of the seven months (January to August, 1924) that he was employed.

Before going to work in this plant he drank heavily, but while employed there he drank considerably less. He dates the onset of his symptoms from July, 1924. The first complaints were general weakness and slowing up of the gait, lethargy, dribbling of saliva and hanging of the lower jaw. His gait became progressively worse, and he noticed that he was dragging the left leg. Shortly after this, he became very nervous. His expression became silly; he developed the typical causeless laughter and a monotonous speech. Later, there appeared an intention tremor of the hands, propulsive gait and retropulsion on standing, and frequent long inspiratory sighing. He also developed numbness and tingling in the arms, hands and feet. The general physical examination was negative except for large, infected tonsils.

Neurologic Examination.—There was slight sway in station, awkward gait with spasticity of the right leg, and a rising movement of the right heel. His posture was slightly stooped. There was exaggeration of all the deep reflexes and increase in muscle tone in both legs. No pyramidal tract signs or ataxia could be elicited. There was a slight intention tremor of both hands. The speech was slow, was monotonous and at times suggested the bulbar variety. The pupils showed no changes, and there was no diplopia or nystagmus. The fundi,

vision fields and cranial nerves were normal. There was a loss of the finer movements of the hands, which was particularly noticeable in writing.

CASE 3.—*History.*—A man, aged 28, night superintendent of a manganese grinding plant, was in more or less constant contact with the dust for about ten months. The family history was negative. The past history showed nothing of importance except that the patient had a mild case of influenza in 1918 and three attacks of quinsy. He had overindulged in the use of alcohol and drank heavily before working in the manganese mill, but during that time he drank considerably, though less heavily.

He dated the onset of his symptoms to August, 1924, at which time there was a slowing up of his speech. He had been working in manganese for about eight months. He then developed uncontrollable, causeless laughter, then slowing up of his gait and dragging of the feet, generalized muscular weakness, particularly in the legs, and a sense of retropulsion on arising. He became nervous and irritable, and had flare-ups of temper and a frequent sighing noise made with long inspiration. There was dribbling of saliva and drooping of the lower jaw. The general physical examination was negative except for infected tonsils and slight thyroid hypertrophy.

Neurologic Examination.—The patient showed a definitely masked expression. His station was steady, the gait was slow and awkward with a tendency to propulsion, and there was some retropulsion on arising. The deep reflexes were all exaggerated. There were no pyramidal tract signs, but the muscle tone was generally increased, the posture was stooped and the finer movements of the hands were clumsy. There was a slight intention tremor of the hands and a coarse tremor of the lips and lids. The lower jaw tended to droop. Speech was slow and monotonous, and enunciation was poor. The pupils showed no change from the normal; there was no diplopia or nystagmus. The fundi and vision fields showed no changes, and the cranial nerves were intact. Mentally, the patient revealed no memory defect; he was somewhat emotional, as shown by irritability and causeless laughter and lack of sociability. There were no other mental symptoms.

CASE 4.—A boy, aged 17, with a negative family history and an unimportant past history which showed no diseases other than mild influenza in 1918 and occasional tonsillitis, said he had drunk a considerable amount of whisky during the time he was working in manganese; otherwise his habits were good.

The patient dated the onset of his illness to July, 1924. He had worked in the manganese grinding plant since January of the same year. The first symptoms noticed were lethargy and difficulty in using the legs, with marked weakness over the entire body, especially in the lower extremities. There developed staggering in walking, some headache, and the personality changes of irritability, causeless laughter and mental exhilaration with verbosity. He became nervous, had outbursts of temper, and developed a frequent sighing noise on long inspiration. Later, his gait became more involved, and there was a propulsion in walking and retropulsion on arising. There were marked tremors of the extremities, which were increased on intention, and he developed paresthesias of the extremities. A general physical examination was negative except for infected tonsils and teeth.

Neurologic Examination.—The patient had slight sway in station, slow, awkward gait and a tendency to stagger. The deep reflexes were exaggerated, and there was a general increase in muscle tone. The finer movements were clumsy; this was especially noticed in writing. There was head nodding and tremor of the legs, face and tongue and some intention tremor of the hands. There was no masked expression. Speech at the time of examination appeared normal, but his mother described a slow, monotonous speech formerly. The pupils showed no changes, and there was no diplopia or nystagmus. The eye grounds exhibited marked pallor of the optic disks, especially of the temporal halves; this appeared to be more than the normal temporal pallor. On rough examination, the vision fields showed no changes. There was suggestive left facial weakness; otherwise, no cranial nerve involvement was noted. Mental examination revealed some emotional changes, with irritability and causeless laughter. The patient was also slightly elevated, though somewhat asocial.

CASE 5.—*History.*—A man, aged 47, married, and the father of three healthy children, gave a negative family history, and had never been sick prior to the time he came in constant contact with manganese dust in this grinding plant. He began work in January, 1925, and worked there three months. He wore a mask part of the time but stated that it was uncomfortable and he did not feel it necessary to protect himself.

The patient's habits were very good. He never indulged in the use of alcohol. He noticed within fifteen days after going to work in this plant that he would stagger forward or backward, and when sitting would have a sensation of falling backward and on arising would have slight retropulsion. He became nervous, irritable and, at times, happy, jovial and slightly elevated. He noticed that he became weak and would tire easily; his memory was impaired, and there were some paresthesias of the hands and feet. A general physical examination was negative except for pyorrhea alveolaris.

Neurologic Examination.—The patient had a slight sway in station, unsteady gait with propulsion in walking, and some retropulsion on rising. The deep reflexes were present but not exaggerated, and there were no pyramidal tract signs, no change in muscle tone, no incoordination. The skin reflexes were active and equal; there was no tremor and no sensory change. No change in speech was noticed, but there was a silly facial expression. The pupils were entirely normal, as were the fundi, vision fields and cranial nerves. Mentally, the patient had recently begun to show some suspiciousness of his wife and children, his memory became impaired, and he showed a slight elevation, silly conduct, a happy, carefree expression and causeless laughter. He was decidedly more jovial and playful than when in his usual health, for his wife stated that he was ordinarily very quiet.

CASE 6.—*History.*—A man, aged 33, married, and with one healthy child, had an entirely negative family history. He had never been sick, but had always been a very heavy drinker. Some years ago, the patient had worked in a manganese grinding plant but did not develop any symptoms at that time. After he was discharged from the army, he again worked for three months in a manganese grinding plant but had to stop on account of illness. He was so mentally abnormal at the time of discontinuing this occupation, and was drinking so heavily that he was committed to one of the state hospitals with the diagnosis of alcoholic psychosis. He was kept in this institution for two months and showed little improvement but was discharged.

He complained of stiffness and vague pains all over his body. The general physical examination was negative.

Neurologic Examination.—The patient showed a steady station; his posture was bent forward and his gait was slow and awkward; the deep and superficial reflexes were present and equal; there were no pyramidal tract signs, no cranial nerve involvement, no sensory changes and no incoordination. There was a marked anteroposterior head nodding, but no tremors of the extremities. There was a generalized muscular weakness without atrophy. The pupils were normal, the eye grounds showed a very reddened vascular retina with no changes in the disks. He had marked lack of expression with a more or less constant silly, causeless laughter. Mental examination showed the patient to be irritable and suspicious and at times, unruly and destructive. His speech was slow and monotonous, and he expressed himself very poorly. His memory was definitely impaired, and he was very asocial. Evidently there was present rather marked mental deterioration.

LABORATORY FINDINGS

It was not possible to obtain laboratory examinations in one of the six cases. A spinal fluid examination was made in each of the others. The cell count was above 10 in two: 12 and 14 cells per cubic millimeter. The globulin was normal, as were the pressures, colloidal gold curves and the Wassermann tests.

The blood Wassermann reaction was negative in all five cases. The spinal fluid and stools showed no trace of manganese in any case. The routine urine examinations were negative, but manganese was detected in the urine in three of the five specimens examined. In two of them, there was only a trace, but in one it was found in abundance, 5 mg. per liter. Manganese might have been found in each case had a larger quantity of urine been examined. In these examinations, 2 liters were used. The patient in whose urine manganese was found in the greatest quantity had discontinued work in the manganese dust two days before he was examined. The two patients in whose urine none of the element was found had been longest away from exposure. No examination of blood smears was made, but no changes in the size or shape of the red blood cells or material variation from normal of the white cells were found by a colleague who did make such a test in one of the cases here reported during the time the patient was most acutely ill.

It is generally agreed that manganese is eliminated chiefly in the feces and to a lesser extent in the urine. Wagner,[13] von Jaksch,[7] Seiffer [5] and all other investigators report no manganese in the urine of their clinical cases even though a careful search was made. In experimental studies on animals, Harnack [14] found only a minute amount of manganese in the urine and concluded that it was eliminated only in a small degree by the kidneys. Cahn [15] is also of the opinion, based on animal experimentation, that manganese is eliminated to some extent by the kidneys, but he found in no case the amount eliminated in this way proportionate to the amount introduced into the body.

COURSE AND PROGNOSIS

In persons who are susceptible to the invasion of the central nervous system by this poison, there appeared to be a gradual progression of the symptoms as long as they continued to absorb the manganese dust. The symptoms remained at the maximum for varying lengths of time and then tended to show gradual improvement up to a certain point. I am of the opinion, because of the well grounded belief that there is a destruction of cerebral tissue causing the symptoms of this condition, that there will be, except in the mildest cases, permanent disability of those poisoned by this metal.

PROPHYLAXIS

Prevention is a problem of the industrial hygienist rather than of the neurologist. Nevertheless, poisoning by the inhalation of manganese dust can certainly be prevented, provided very simple precautions are rigidly maintained. No face mask through which a man must breathe while at work is pleasant to wear, but, because of the dangers encountered, the wearing of an approved mask should be rigidly enforced. The exposed skin of men working in the mill which I inspected was literally caked with manganese; no gloves were worn, and the ore was thick under the nails. It is hardly conceivable that men working in this mill are very different from other mill workers, and, if this is true, we know that their personal hygiene is not all that could be desired. Therefore, the installation of showers, and insistence on the part of the mill operators that baths be taken and a change of working clothes be made at the end of each day's work is essential. A very important and necessary preventive measure would be an efficient fan-suction dust collector which would keep the air in circulation, collect a large part of the dust and thus lessen the chance of poisoning.

TREATMENT

The treatment suggested for the condition has been massage, corrective exercises, warm baths and various types of hydrotherapy, together with stimulation of the excretion of the poison by general elimination.

SUMMARY

1. Manganese, more often than is recognized, causes symptoms in workmen handling this ore.

2. That certain persons are not susceptible to the poisoning effects of manganese is demonstrated by the fact that many workmen in this plant have been in contact with manganese dust for several years with no apparent ill effect.

3. Mental symptoms have been described by some investigators and denied by others. Mental changes were found in each of the patients of this series.

4. No record could be found of other investigators having detected manganese in the urine in clinical cases. Experimentally, it had been found in minute amounts. In this series, it was present in three of the five specimens of urine examined.

212 West Franklin Street.

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14. Harnack, Erich: Arch. f. exper. Path. u. Pharmakol. **46**: 372, 1901.
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ARTHROPLASTY OF THE TEMPORO-MAXILLARY JOINT *

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To state briefly the anatomy of the temporomaxillary joint and its associated parts, first, from the osseous standpoint, there is the condyle and its neck, the sigmoid notch and the coronoid process. Overlying these structures, we have the zygomatic process and its anterior root, which is broad and strong, to terminate in a rounded eminence, the eminentia articularis. This eminence forms the anterior boundary of the glenoid fossa, and in the recent state is covered with cartilage. The middle root forms the posterior boundary of the mandibular portion of the glenoid fossa. The glenoid fossa is bounded posteriorly by the vaginal and auditory processes. The junction of the anterior root with the zygoma is a projection called the tubercle, for attachment of the external lateral ligament of the lower jaw. Between the anterior and middle roots is an oval depression forming part of the glenoid fossa for the reception of the condyle of the lower jaw. This fossa is bounded in front by the eminentia articularis; behind, by the tympanic plate and the auditory process, and is divided into two parts by a narrow slit, the gasserian fissure. The anterior or mandibular part, formed by the squamous portion of the bone, is smooth, covered in the recent state by cartilage, and articulates with the condyle of the lower jaw. This part of the glenoid fossa is separated from the auditory process by the postglenoid process, which, in mammalia, prevents backward displacement.

The ligaments that enter this joint are the external lateral, the capsular, and the interarticular fibrocartilage.

* Read before the Section on Stomatology at the Seventy-Sixth Annual Session of the American Medical Association, Atlantic City, N. J., May, 1925.

Overlying this bony anatomy, the important structures that must be guarded are: of first importance, the frontal and orbital branches of the facial nerve, the temporal artery and its branch, the transverse facial, the temporal vein and the auricular temporal nerve. Immediately internal to the condyle is the internal maxillary artery, which is injured occasionally when the head of the condyle is removed. The temporal artery and vein, with its branches, occasionally gives a most troublesome hemorrhage when torn or cut unintentionally; but, generally speaking, the larger branches can be avoided. It is the frontal and orbital branches of the facial nerve that are frequently severed in this operation, and, because of the uncertainty of their position, I suggest the vertical incision. I am sure that most operators have encountered the occasional case in which the frontal if not the orbital branches have been divided. This is not to be mistaken for the paresis shown in the inability to close the upper eyelid on that side following operation, which is due to the retraction during the operation. Here I would suggest that the mastoid self-containing retractor be used.

HISTORY

Briefly, the history is as follows: Humphrey reports resection of the head, in 1854; Grube, in 1863; Whitehead, in 1874, and König, in 1878. The operation has been modified by different surgeons. Helferich interposed muscle from the temporal; Mikulicz interposed masseter muscle, and Murphy used fascia from the temporal muscle. Roser interposed a gold plate; Payr suggested magnesium; Hofmann used free periosteum; Rochet and Poisson resected the ascending ramus. Many other operations have been suggested.

ETIOLOGY

The sexes are about equally affected. Orlow found sixty-eight patients (about 80 per cent.) between 1 and 10 years; 15 per cent. between 11 and 20; 5 per cent. between 21 and 30. Blair gives from 2 to 57 years, 25 per cent. of whom were under 10. In Orlow's cases, trauma was the cause in 29.4; otitis media, either primary or as a complication of some general infectious disease, 22 per cent.; osteomyelitis, periostitis of neighboring bones, 12.6 per cent.; rheumatic, gonorrheal and other inflammation of the mandibular joint, 10.5 per cent. Blair reports that 50 per cent. of his cases were due to trauma. In my series of seventeen cases, one was due to diphtheria, two were congenital and fourteen were due to trauma.

In considering this subject, I am following the classification of articular, extra-articular and articular-extra-articular. Three of my series were extra-articular and the others articular. I believe that a modified incision is much safer, and that in some cases of extra-articular trismus the epithelial inlay suggested by Esser in 1916, and modified by Waldron in 1917, at Sidcup, England, is most useful. I have searched the literature, and believe the method I am reporting is new, if not original.

OPERATION

The usual incision is L-shaped, the vertical limb extending from the level of the floor of the external auditory meatus and parallel with the ear but anterior to the temporal artery, and the horizontal or short limb extending forward slightly above the zygoma for about 2 cm., but only through the skin. I suggest that, because of the difficulty of locating with precision the orbital branch of the facial, which is frequently divided in this operation, only the vertical incision be made, about 1 inch long, starting from the level of the floor of the external auditory meatus. I fully recognize that this gives a poor exposure, but with patience, the condyle and even the coronoid may be removed through this incision. On removal of the condyle, I have filled the glenoid area with free fascia lata from the hip, and have also used Cargile membrane; but I prefer the former.

The value of any interposed tissue is open to doubt, as considerable experimental work has been done, more particularly with fat, which has disappeared in a very short time—in my own cases, within less than two months' time. I do believe that the holding apart of the remains of the ascending ramus and the cup-shaped cavity (the glenoid fossa) is important.

The shaping of the ascending ramus from which the head of the condyle has been removed is of great importance. It should be pointed anteroposteriorly and narrow laterally. By that, I mean that the new head should be diamond-shaped, and as narrow or thin as possible, in this way allowing for a very small attachment of fibrous tissue.

The case I wish to report in particular is one in which the trismus was due to a fibrotic change in the internal pterygoid muscle, due to shrapnel. The muscle was largely replaced by fibrous tissue, and the mouth could be forced open under a general anesthetic but would return to the fixed position in a short time. It was decided that an epithelization of that area would overcome the contraction due to the fibrous tissue. An epithelial inlay was implanted near the attachment of the internal pterygoid muscle through an incision parallel with the acclusal surface of the lower teeth. Dental compound was molded to fit in this incision, which had been carried well through the fibrous tissue and held in position by silkworm-gut sutures. It was necessary to implant three inlays before the mouth could be opened to its fullest extent.

The most satisfactory results we have obtained were in cases of extra-articular trismus due to the union of the coronoid process to the inner surface of the zygoma or to the base of the pterygoid process of the sphenoid. This was, of course, of traumatic origin. In these cases, the coronoid process was resected but the joint was left intact, and the result was excellent.

There is another feature of these cases that should not be neglected; that is, the recession of the chin. It has been suggested that a double resection of the ascending ramus be made and that the lower jaw be pulled well forward and held in this position by attachment to the upper jaw. This, I feel, is an excellent method if it can be used, but I have found it difficult to adapt this treatment for a number of my cases. I would suggest the following:

A seventh or eighth rib of the proper curvature and length should be taken from the patient and inserted over the mental process of the lower jaw. It is necessary in these cases to roughen the outer surface of the mandible and the inner surface of the graft, and wire it into position with any light wire, say the medium orthodontia wire. Needless to say, the technic of this operation is very exacting, as it is a free transplant, and in the dissection, one must not go too far up on the outer surface of the lower jaw, for fear of entering the mouth through the neck incision.

CONCLUSION

These cases, while they appear simple because the joint is superficial, present many difficulties, and the final results are not so uniform as one may desire; but in my short experience, by following the procedure outlined in this short paper, we were able to obtain even in the worst cases a moderately good result; consequently, I believe in the operation as near the joint as possible.

ABSTRACT OF DISCUSSION

DR. T. E. CARMODY, Denver: For a number of years I have used the incision Dr. Risdon recommends, and I can obtain plenty of opening through this incision for the removal of the condyle. As Dr. Risdon says, it is difficult at times because the opening is small, but by use of the mastoid retractor for forcing back the tissues, one can, as a rule, get sufficient opening for removal. The fact that paralysis does not develop or that one is not quite so liable to injure the temporal artery or the internal maxillary artery makes it worth while to take a little extra trouble. It prolongs the operation only a few minutes, but not enough to interfere with the tissue. For some years, I have not placed the tissue in between the parts of the mandible, and I find that my results are just as good, especially as there is such a small amount of tissue.

DR. ROBERT IVY, Philadelphia: I believe that Dr. Blair of St. Louis, in 1911, was the first one to advocate the interposition of temporal fascia, even before Dr. Murphy. The incision that Dr. Blair employs is most suitable when it is desired to use temporal fascia. The incision ascends vertically in front of the ear, then curves forward well above the zygoma. We do frequently get some paralysis of the occipital frontalis muscle, but I never have seen paralysis of the orbicularis palpebrarum.

DR. HERBERT A. POTTS, Chicago: I should like to point out the dangers of wholesale extraction of teeth. The extraction of a tooth requires as much care, attention and thought as the opening of the abdomen, because of the complications that may follow. A young woman had three third molars extracted. Whether or not they were infected at the time or whether they became infected afterward I do not know, but, at any rate, she developed a pyremia, abscesses in each groin, a pleural abscess and an abscess in each parotid, from which she almost died. The parotid abscesses resulted in a complete bony ankylosis of both temporomandibular joints. The jaw was just plastered to the base of the skull from the joint clear out to the tip of the coronoid process. An incision was made similar to the one which Dr. Ivy showed, and the mandible was liberated from the base of the bone, but on one side, just after liberating the coronoid process, I opened a vein and a severe hemorrhage resulted. The wound was perfectly clean for a week, with only a provisional drain placed in it; a week later in operating on the other side, I made the mistake of opening the first wound and placing some fascia lata in at that time. On the left side one can hardly see the line of incision through which this operation was done. I think that, had I refrained from opening the first wound, I should not have had the slight infection and the scar on the other side. But the principal danger is in extracting a tooth, dismissing the patient, and not giving any further attention to him.

DR. E. FULTON RISDON, Toronto: The interposition of material certainly is not necessary. I never take off the zygoma; it leaves things projecting in a rather unsightly way. If one takes off a section of the zygoma, of course, one must bring it over; it cannot be brought under. These operations should not be undertaken lightly; especially should the incision be kept high, never coming below the level of the floor of the external auditory meatus. A number of patients have come to me recently with a total paralysis of the muscles on that side; it is a question whether these anastomoses are very successful.

Gastro-Enterostomy.—Of all the operations for benign lesions of the stomach or duodenum, gastro-enterostomy has the widest range of usefulness. When indications are adequate, gastro-enterostomy leaves little to be desired.—Balfour.

THE TREATMENT AND PROGNOSIS IN MYXEDEMA *

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When Dr. George Murray [1] read his classic paper before the British Medical Association in July, 1891, on the treatment of myxedema by the administration of the thyroid gland of sheep, the medical world in general proclaimed, and quite correctly, that there had been discovered another specific remedy for what had usually been regarded as a hopeless condition. Marvelous as the cure may be in most instances, it is obvious to any one who has observed a group of these patients under treatment over a long period, that even though the diagnosis is correct and the preparation used in treatment is potent, there is often a failure, either partial or complete, to effect a cure. This may be due to several reasons which will be considered later, but undoubtedly the chief cause is the careless administration of a drug which must be given with the greatest exactness in order to produce satisfactory results. It is often apparent that mistakes in the management of these patients have been made because the treatment of myxedema has been considered too simple.

It is our purpose in this study to appraise carefully the method of treatment that has been used on a series of patients in this hospital in an endeavor to formulate a method whereby treatment can be administered with the greatest efficiency and safety. In addition, an opportunity has been afforded to observe a number of patients over a considerable period, and certain conclusions concerning the prognosis in this disease have been formulated.

This study is based on a series of twenty-six patients, all of whom presented the typical symptoms and signs of myxedema and, in addition, all had a characteristically diminished basal metabolism. A fairly large group of patients was excluded from the series as the diagnosis in some instances was in doubt or the condition atypical in various aspects. It is fortunate that the same preparation of dried thyroid gland (Armour's) has been used since the hospital was opened, and there has never been any conclusive clinical evidence that the potency of the drug has varied. There may be a great variation in the strength of the drug as prepared by different manufacturers according to the careful studies of Dr. Reid Hunt,[2] who has been interested in this important question for a good many years. In his opinion, this variation in potency results from the fact that there is no generally accepted standard for thyroid. At present, the most sensible procedure for the clinician is to use one preparation constantly and determine by careful clinical observations its strength and dosage. Treatment was controlled in all the patients of this series by (1) the clinical response of the patient, (2) the basal metabolism, (3) the resting pulse rate, and (4) the body weight. Data concerning this group of patients are shown in the accompanying table. No attempt will be made in the present paper to discuss the clinical aspects of myxedema or the relation of the low metab-

* From the Medical Clinic of the Peter Bent Brigham Hospital.

1. Murray, George: On the Treatment of Myxedema by the Hypodermic Injection of an Extract of the Thyroid Gland of the Sheep, Brit. M. J. **2**: 796 (Oct. 10) 1891.

2. Hunt, Reid: Standardization of Thyroid Preparations, Arch. Int. Med. **35**: 671 (June) 1925.

olism to the disease, as these features have been reported from the medical service of this hospital in a previous communcation.[3]

METHOD OF ADMINISTERING THYROID GLAND

It has long been emphasized that the most satisfactory method of treating myxedema is to divide the therapy into two stages, (1) the initial dosage, which is calculated to restore the patient to a normal level of metabolism, and (2) the maintenance dosage, which is the smallest amount necessary to keep the metabolism within normal limits. For convenience, our experience in the management of each stage of the therapy will be considered separately.

In untreated patients with the characteristic picture of myxedema, we have found that the amount of dried thyroid gland which is necessary to restore the basal metabolism from the initial low point to normal has varied widely, as the total amounts

Data in Thyroid Treatment of Twenty-Six Patients With Myxedema

	Age	Sex*	Initial Metabolism Before Treatment; per Cent. of Normal	Duration of Symptoms	Maintenance Dose, Daily
1. G. L. F.	22	♀	—34	2 yr.	0.13 gm.
2. E. R. F.	22	♂	—29	2 mo.	0.13 gm.
3. J. F. K.	50	♂	—25	1 yr.	0.13 gm.
4. M. H.	50	♀	—28	6 yr.	0.13 gm.
5. M. T. M.	73	♀	—33	28 yr.	0.13 gm.
6. D. McK.	43	♂	—40	6 yr.	0.195 gm.
7. J. F. B.	48	♂	—32	3 yr.	0.086 gm.
8. S. H. V.	42	♂	—38	18 mo.	0.13 gm.
9. T. B. F.	42	♀	—34	7 yr.	0.13 gm.
10. A. A.	56	♂	—26	1 yr.	0.13 gm.
11. S. E. P.	64	♀	—26	2 yr.	0.13 gm.
12. E. M. S.	67	♀	—26	5 yr.	0.195 gm.
13. M. J. B.	62	♀	—49	2 yr.	0.065 gm.
14. K. O.	50	♀	—41	6 yr.	0.13 gm.
15. F. M. W.	50	♀	—36	17 yr.	0.195 gm.
16. F. N. T.	60	♂	—39	7 yr.	0.097 gm.
17. A. L. A.	52	♀	—28	5 yr.	0.13 gm.
18. C. M.	38	♀	—31	3 yr.	0.195 gm.
19. J. H. T.	33	♂	—36	3–5 yr.	0.13 gm.
20. M. J. S.	53	♀	—33	4 yr.	0.195 gm.
21. A. E.	49	♂	—18	4 yr.	0.39 gm.
22. L. B. C.	39	♀	—24	.....	0.195 gm.
23. H. T. B.	59	♀	—36	4 yr.	0.195 gm.
24. B. A. M.	52	♀	—31	8 yr.	0.13 gm.
25. J. C. F.	40	♂	—26	3 yr.	0.030 gm.
26. P. W.	41	♀	—18	3 yr.	0.065 gm.

* In this column, ♂ indicates male; ♀, female.

necessary in this series have ranged from 2.3 to 6.5 gm. This variation has been due, in part at least, to the lack of a constant time interval over which the drug has been given. For example, in some patients the metabolism has been raised to normal by relatively large doses which were given for a few days, while in others the same result has been obtained by the administration of much smaller doses for a longer period. Future studies with a uniform dosage may yield more constant results. Several years ago, it was thought that it might be possible to estimate precisely the amount of dried thyroid necessary, from the patient's body weight and the degree of the change in the metabolism in a similar manner to the modern method of calculating digitalis dosage, in patients with heart disease, from the body weight. Our present experience has suggested, however, that sex, age, body weight, duration of the disease, severity of the symptoms, and the initial level of the metabolism bear no accurate or constant relationship to the amount of thyroid gland necessary to restore the patient to a normal condition.

3. Sturgis, C. C.: A Clinical Study of Myxedema, with Observations on the Basal Metabolism, M. Clin. N. Am. 5: 1251 (March) 1922.

While it may be possible to calculate the exact effect of a definite dose of thyroxin on a myxedematous patient when it is given intravenously, we have not been able to determine the effect of a known amount of dried gland with a similar degree of accuracy when given by mouth. This might be expected, however, as thyroxin is a highly refined preparation of known chemical composition, while "dried thyroid gland" is of a less definite nature and, moreover, can be given only by mouth. This, therefore, introduces the complicating factor that the drug may not undergo complete absorption in the gastro-intestinal tract. The failure to formulate a precise method of estimating the amount of dried thyroid gland to use in the initial stage has led to the adoption of a known safe dosage which is given over a variable length of time depending on its effect on the patient. In the average adult patient without complications, it has been found that a dosage of 0.13 gm. (2 grains) three times a day can be continued with safety until the basal metabolism and pulse reach normal.[4] With this dosage, the metabolic level and pulse rate usually reach normal in from five to ten days, although individual patients show a wide variation in this respect, and it is advisable, therefore, to keep the patient under close observation and continue with this amount until satisfactory results are obtained. In treating patients with uncomplicated myxedema, it is more satisfactory to confine them in a hospital for from two to three weeks, as this permits close observation and their activity can be controlled. As profound changes occur in the patient's metabolism during the early stages of treatment, it is desirable to keep them in bed for at least a week as a precaution against unpleasant symptoms.

If the patient is under observation in a hospital, the initial dosage of 0.13 gm. (2 grains) three times a day is continued until the metabolism and resting pulse rate approach normal and there is clinical evidence of improvement. It has been our custom to determine the basal metabolism, resting pulse rate, and body weight at least every other day in order to control the dosage more accurately. After thyroid gland has been given in amounts of 0.13 gm. three times a day for three or four days, the first clinical evidence of a thyroid effect is shown by the patient experiencing a sensation of increased warmth, moderate sweating and accelerated mental reactions. Coincident with these changes, there is usually a slight increase in the resting pulse rate, averaging about 10 beats a minute, and, likewise, the basal metabolism shows an average rise of 10 per cent. At this time, the patient's temperature may become somewhat elevated above normal for a few days, the average, however, not exceeding 100 F. As untreated patients with myxedema frequently have a subnormal temperature, this change following treatment is one of considerable magnitude, and it is not uncommon for a patient under these circumstances to complain of malaise, mild generalized aches and pains, and loss of appetite. The drug should be continued, however, until the basal metabolism is between — 10 and — 5, according to the standards of Du Bois. While it is generally accepted that the normal varies between — 10 and + 10 according to these standards, it has been our experience that the average patient with myxedema more nearly approaches a condition approximating normal if the metabolism is maintained between — 5 and — 10. If the metabolism remains at this

4. The same amount may be given four times a day if the patient is under observation in a hospital and no complications exist.

level for five or six weeks, there will be a loss of weight which averages from 10 to 15 pounds (4.5 to 6.8 kg.). This loss is due apparently to the disappearance of the subcutaneous, nonpitting edema that is characteristic of the disease. After several months of treatment, it is commonly observed that the patients regain this weight and 5 or 10 pounds (2.3 or 4.5 kg.) in addition, which doubtless results from their greatly improved general nutrition.

After the patient's basal metabolism and pulse rate have been restored to normal and there has been evidence of clinical improvement, the second stage of treatment is begun. The object at this time is to determine the exact dosage which will maintain the metabolism at the proper level. This has been called the maintenance dose, and is the amount that is to be taken by the patient each day. This dosage when once established is usually constant and in most instances must be continued throughout a patient's life, although occasionally the size of the dose must be altered, and in very rare instances it appears that there may be complete recovery from myxedema, as the drug may be discontinued without a recurrence of the disease. Our experience has taught us that most frequently the correct dose is in the vicinity of 0.13 gm. (2 grains) a day, although in individual patients the amount has varied from 0.065 gm. (1 grain) to 0.195 gm. (3 grains) daily. In a study of twenty-five patients with myxedema who have been observed over a sufficient length of time to make our results significant, we found that fourteen required a maintenance dose of 0.13 gm. (2 grains) daily; six, a dose of 0.195 gm. (3 grains); two, a dose of 0.065 gm. (1 grain), and three, a dose of 0.098 gm. (1.5 grains). Occasionally a patient has taken as much as 0.39 gm. (6 grains) as a maintenance dose for a short period, but this amount has invariably produced symptoms of overdosage if continued for any length of time.

It is possible to carry out the entire treatment in the patient's home, although the results are less satisfactory, as the dosage cannot be regulated with great accuracy. Moreover, a longer time is required to restore the patient to normal, as the initial dosage must be smaller in order to avoid the possibility of undesirable symptoms. After the diagnosis has been established, if it is desired to treat the patient at home, the following plan has been found to be satisfactory: Dried thyroid gland is given in a dosage of 0.13 gm. (2 grains) three times a day for five days. During this interval, the patient should be confined to the house and should spend a major part of the time resting in bed. Each day the pulse rate should be counted by the patient or by some member of the family, before the patient arises in the morning. In this way the basal pulse rate is obtained, and such factors as food and muscular activity, which influence the pulse rate, are eliminated. In the absence of basal metabolism estimations, a careful determination of the resting pulse rate is of great value as it gives an accurate idea of the basal metabolism in most instances. This relationship has been emphasized in a previous communication[5] from this hospital and has been confirmed by others.[6] Under certain conditions, however, such as complete heart block, the pulse rate may remain unchanged despite an increase in the basal metabolism.

Such an occurrence is illustrated by the case record of a patient reported by Aub and Stern.[7] A young woman with a complete heart block and a pulse rate which varied from 49 to 63 a minute was given large doses of thyroid substance. Although the basal metabolism increased to + 40, there was no coincident changes in the pulse rate. Such a possibility, although rare, should always be considered if the pulse rate is used as a guide to the administration of thyroid gland. The body weight should also be obtained before the treatment and again at the end of five days. If the resting pulse rate has not increased above 80 a minute and there has been a loss of only 2 or 3 pounds (0.9 or 1.4 kg.) in body weight, there is no indication of overdosage, provided the patient does not complain of palpitation, dyspnea, excessive warmth, dizziness or nausea. If all is satisfactory, the treatment may be continued by giving the thyroid tablets in amounts of 0.13 gm. twice a day. This dosage should be maintained until the resting pulse rate is in the vicinity of 75 a minute, unless symptoms of overdosage appear. When the pulse rate reaches a satisfactory level, the amount of thyroid gland that is given should be reduced to 0.13 gm. daily, and this should be considered as the maintenance dose. A determination of the basal metabolism at weekly intervals is very helpful in regulating the treatment.

The chief difficulties that are encountered in the management of patients with myxedema may for the most part be attributed to careless observation on the part of the physician or failure of the patient to follow instructions. If a patient with myxedema is given a prescription for thyroid tablets and instructed to take a certain number each day and report back in a few weeks, the possibility of an underdosage or overdosage is likely. The latter more commonly occurs with the result that unpleasant and possibly serious symptoms develop. This is unfortunate as the patient is frequently reluctant to continue with further treatment, since in most instances following an overdosage there develops an abnormal fear of the drug, and a tendency to attribute every possible symptom that develops thereafter to an excess of the thyroid substance.

An additional difficulty in the treatment of myxedema is the lack of cooperation of the patient. This is shown by the observation that approximately one fourth of our patients have at some time during the course of their treatment discontinued taking thyroid tablets of their own volition, despite our instructions. As a result they have had, with a very rare exception, either a partial or a complete relapse of the disease. This lack of cooperation results from a failure of the patients to appreciate the importance of continuing with the drug after all symptoms of the disease have disappeared. After the drug has been omitted for a period of several weeks, the condition of partial or complete myxedema again becomes apparent, with a rare exception. Following this, the characteristic mental dulness and lack of interest become manifest, and the patient no longer has the initiative to consult a physician. On account of the tendency of so many to discontinue treatment, we have for the last three years required all patients with myxedema to report to the outdoor department at regular intervals for observation, in order to eliminate this important cause of therapeutic failure. At each visit a careful examination is made, and if there is any indication that the optimal

5. Sturgis, C. C., and Tompkins, Edna H.: A Study of the Correlation of the Basal Metabolism and Pulse Rate in Patients with Hyperthyroidism, Arch. Int. Med. **26**: 467 (Oct.) 1920.

6. Read, J. M.: Correlation of Basal Metabolic Rate with Pulse Rate and Pulse Pressure, J. A. M. A. **78**: 1887 (June 17) 1922. Minot, G. R. and Means, J. H.: The Metabolism-Pulse Ratio in Exophthalmic Goiter and Leukemia, Arch. Int. Med. **33**: 576 (May) 1924.

7. Aub, J. C., and Stern, N. S.: The Influence of Large Doses of Thyroid Extract on the Total Metabolism and Heart in a Case of Heart-Block, Arch. Int. Med. **21**: 130 (Jan.) 1918

treatment is not being carried out, the basal metabolism is determined and the dosage is adjusted accordingly. If it becomes necessary to adjust the maintenance dosage, the patients are required to record their resting pulse and body weight daily over a period of several weeks. These data, when considered with a careful inquiry into the symptoms and combined with the basal metabolism determinations, eliminate careless observation and afford an accurate guide for the proper regulation of treatment.

SERIOUS COMPLICATIONS THAT MAY BE ENCOUNTERED IN TREATING PATIENTS WITH MYXEDEMA WITH THYROID GLAND

There are several complications that may exist in patients with myxedema which make the administration of thyroid gland a somewhat hazardous procedure unless the treatment is very carefully controlled. The complications involve chiefly the heart, kidneys and blood and are observed more frequently in the middle aged or elderly patients

Recently, Fahr [8] has emphasized that in myxedema there may be definite symptoms and signs of heart failure which do not respond to the therapy of rest and digitalis but are cured by thyroid medication. Fishberg [9] has likewise pointed out that a greatly dilated heart occurs commonly in myxedema and, furthermore, a generalized arteriosclerosis may be observed even in young patients. The latter reported the case of a man, aged 21, with myxedema, in whom necropsy showed an enlarged heart and, in addition, a generalized arteriosclerosis, which included atheromatous patches of the arches of the aorta, was present. Swan [10] has reported his observations on a patient with myxedema in whom auricular fibrillation appeared following the use of thyroid substance. The rate became regular when the drug was discontinued; auricular fibrillation reappeared on renewal of the treatment and disappeared a second time when the drug was withdrawn. The point we wish to emphasize is that patients with myxedema may have a serious impairment of the myocardium, and this may be improved or cured by the administration of thyroid gland; but under these circumstances, the drug should be given with great caution as otherwise unfavorable and fatal results may follow. This is well illustrated by the following case report of a patient who was recently observed in the Peter Bent Brigham Hospital:

M. H. B., a Scotch woman, married, aged 50, entered the hospital, Feb. 17, 1925. She gave the classic history of myxedema extending over a period of several years, and this was confirmed by the physical examination and a basal metabolism of —32. In addition, the patient gave a history of angina pectoris and, furthermore, showed a severe anemia as indicated by a red blood count of 2,000,000 cells per cubic millimeter. Treatment was given in the form of dried thyroid gland in doses of 0.13 gm. (2 grams) three times a day. After six doses were taken, the temperature suddenly rose to 101 F. but returned to normal in a few hours. There was no apparent explanation of the febrile reaction, and as there was no other evidence at the time that the dosage was excessive, it was decided to continue with the thyroid therapy. Accordingly, eight more doses of 0.13 gm. were given in the next twenty-four hours. The basal metabolic rate at this time was found to be —11. The patient's condition, however, suddenly became alarming, as indicated by a weak, feeble pulse of 120 a minute, cyanosis, cold and clammy skin, and a temperature of 101 by mouth. Although the thyroid medication was immediately discontinued, it was a week before the patient recovered from this state of profound collapse. Following this, the thyroid substance was omitted for sixteen days, and during this interval digitalis was administered in full doses. At the end of this time, dried thyroid gland was again given in doses of 0.03 gm. twice a day. Two days later, the patient was found in collapse and pulseless and died in a few minutes without regaining consciousness. Necropsy showed coronary arteriosclerosis, cardiac infarction, hypertrophy and dilatation of the heart, atrophy and fibrosis of the thyroid gland and generalized arteriosclerosis.

This fatality illustrates very strikingly the disaster that may follow the administration of thyroid gland in a patient who has serious heart disease. Apparently the myocardium of this patient was unable to meet the strain imposed on it by the increased cellular metabolism that followed the use of thyroid gland, even in moderate doses. Under the circumstances, there was an increased demand for oxygen by the body tissues, and this could be delivered to the cells only by the blood stream. As a result, it was necessary to augment the circulation rate, and an added strain was thereby put on the heart muscle, which was unable to respond. An added handicap was the frequently observed anemia of the disease [11] as indicated by a red blood count of 2,000,000 cells per cubic millimeter, which resulted in a diminished ability of the blood to carry oxygen. Consequently, the heart was obliged to perform much more work, as each unit of blood carried less oxygen than normal and, therefore, a greatly increased circulation rate was necessary. This experience taught us that a more logical and safer procedure to carry out in such a patient is to give two or three blood transfusions of from 250 to 300 c.c. each and thereby increase the oxygen carrying capacity of the blood. Moreover, such a patient should be thoroughly digitalized. It is also of great importance that the initial doses of thyroid gland should be exceedingly small, such as 0.03 gm. (one-half grain) twice a day. If no untoward symptoms develop, the dose may be cautiously increased.

In addition to changes in the heart and blood, it has long been known that a majority of patients with myxedema have albumin and casts in the urine, and some have a definite renal insufficiency, as shown by the functional tests. The danger that may result under these circumstances is well shown by the studies of Bowen and Boothby [12] which were reported from this hospital in 1917. They found that moderate amounts of thyroid gland by mouth, in patients with chronic nephritis, caused an increase in the blood urea nitrogen and serious effects may result. In one case that they reported, the blood urea nitrogen rose, during the course of a few weeks, from 148 mg. per hundred cubic centimeters of blood to 216 mg. following thyroid feeding. From these studies, it is obvious that all patients with myxedema who are unable to excrete nitrogen in normal amounts, as indicated by an accumulation of nitrogenous products in the blood stream, should be given thyroid gland medication with extreme caution.

It seems advisable, therefore, before giving thyroid gland by mouth to a patient with myxedema, to consider carefully the patient's history, physical examination and laboratory studies, and to ascertain whether

8. Fahr, George: Myxedema Heart, J. A. M. A. **84**: 345 (Jan. 24) 1925.

9. Fishberg, A. M.: Arteriosclerosis in Thyroid Deficiency, J. A. M. A. **82**: 463 (Feb. 9) 1924.

10. Swan, J. M.: A Case of Auricular Fibrillation Occurring During the Administration of Thyroid Substance, Ann. Clin. Med. **3**: 311 (Oct.) 1924.

11. Emery, E. S., Jr.: Blood in Myxedema, Am. J. M. Sc. **155**: 577 (April) 1925.

12. Bowen, B. D., and Boothby, W. M.: A Study of the Effect of Thyroid Medication on the Basal Metabolism, Renal Function and Nitrogen Balance in Chronic Nephritis and in Hyperthyroidism, J. Urol. **1**: 469 (Oct.) 1917.

there are any abnormalities of the heart, blood or kidneys. If complications are present, the patient should be kept strictly in bed and thyroid given in very small amounts. If the heart is markedly involved, digitalis and rest should be given before treatment with thyroid gland. If the patient has an anemia, a transfusion should be considered before the thyroid treatment is begun. If a chronic nephritis exists and there is impaired renal function, the thyroid dosage should be small, the diet reduced in protein, and constant watch should be kept of the blood urea nitrogen.

THE PROGNOSIS IN MYXEDEMA

It is commonly accepted that a patient who is suffering from myxedema can be restored to normal health by the proper administration of dried thyroid gland. It is likewise thought that it is necessary for a patient with myxedema to continue taking thyroid gland substance indefinitely, in order to remain in normal health. While these statements are essentially true, both have had what appeared to be exceptions, in our experience.

The prognosis for cure should also be guarded if the patient shows advanced lesions involving the central nervous system, heart or kidneys. While the general condition may be markedly benefited and the complicating factor may likewise show improvement, it is too much to expect that definite anatomic changes in the heart, nervous system or kidneys can always be completely eradicated. The following record indicates the wisdom of a guarded prognosis in a patient with myxedema and an advanced renal lesion.

A woman, aged 62, had had the characteristic symptoms of myxedema for ten years. Physical examination showed the striking signs of the condition, and, in addition, there was evidence of renal injury as indicated by albumin and casts, and a phenolsulphonephthalein excretion of 8 per cent. in two hours and ten minutes. Following the careful administration of thyroid gland, there was a complete disappearance of all symptoms that could be attributed to the lack of thyroid secretion. In addition, the renal lesion showed some improvement, but the patient died in a few months of uremia.

Serious mental changes are likewise difficult to effect although the general symptoms and signs of myxedema in such a patient may be relieved. The following case record illustrates the inability of thyroid medication to relieve mental symptoms in this condition:

An Irish domestic, unmarried, aged 50, entered the hospital, Dec. 30, 1923, complaining of a gradually increasing loss of strength and energy of six years' duration. The patient had observed dryness of the skin, loss of hair, absence of sweating, and susceptibility to cold for several years. Her memory had failed a great deal, and there had been a very evident mental retardation. The basal metabolism was —28, and the resting pulse rate 54 a minute. Following careful treatment with thyroid gland, the basal metabolism and pulse rate returned to normal, and the general symptoms of myxedema disappeared. After the patient had remained on a maintenance dose of 0.13 gm. a day for several months, she discontinued the thyroid tablets on her own responsibility. Six weeks later, the metabolism was found to be within the limits of normal despite the lack of thyroid medication. Moreover, there was no evidence of a recurrence of the indications of myxedema, such as dry skin, loss of hair, or susceptibility to cold. The mental condition was progressive, and in a short time the deterioration became so marked that it became necessary to admit her to a hospital for treatment of mental disorders. The last basal metabolism, which was normal, was determined after she had not taken thyroid gland for four or five months.

This case illustrates two points of importance in the treatment of myxedema: (1) Serious mental changes which may be associated with the disease are not always relieved by thyroid therapy, and (2) in some instances the metabolism may remain within normal limits for at least four or five months after the thyroid medication is discontinued.

Further evidence that a patient may recover from spontaneous myxedema is furnished by the history of the following patient:

A grocery clerk, married, aged 33, who entered the hospital, Dec. 29, 1916, was found to have a dry, scaly skin, slow speech, retarded mental reactions, and loss of hair. The patient's temperature was subnormal, the resting pulse rate was 71 a minute, and the basal metabolism was —26. Thyroid therapy was instituted and was followed by a striking improvement in the patient's condition. The skin became warm and moist, a regrowth of hair occurred, and the appetite increased. He became more active physically and mentally. Six months later the thyroid tablets were discontinued because the patient felt so well. He was not seen again until October, 1924, eight years later, at which time he appeared at the hospital merely to report how well he had been. During this interval, all medication had been stopped, and the patient had remained in perfect health. The basal metabolism in October, 1924, was found to be +11.

Such evidence that a patient may make a recovery must be accepted with caution and should only be regarded as suggestive, as it is difficult to prove conclusively that the patient was suffering from myxedema when first observed in 1916, although the evidence in favor of this diagnosis is very strong. While these two exceptions have been observed, it can be stated definitely, however, that in almost every instance there is very little tendency toward spontaneous recovery, and that continued therapy throughout a patient's life is necessary for the maintenance of normal health.

DANGERS IN THE USE OF CERTAIN HALOGENATED PHTHALEINS AS FUNCTIONAL TESTS*

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In the last few years, certain halogenated phthalein compounds have come into widespread use as test materials for hepatic function and as mediums for cholecystography. For some time I have been using several of these compounds and have noticed, as a result of the intravenous injection of them into patients, reactions that are, potentially at least, dangerous to the individuals so treated. It is somewhat surprising that, in spite of the rather voluminous literature dealing with these substances, so far little attention has been paid to the untoward reactions following their use. The most important of these compounds has been phenoltetrachlorphthalein, but recently tetrabromphenolphthalein and tetra-iodophenolphthalein have become quite prominent in cholecystography.

Rowntree, Hurwitz and Bloomfield [1] were the first to make clinical use of phenoltetrachlorphthlein as a liver function test. Because of the many difficulties inherent in the measurement of the amount of dye recovered in the feces, it was not long before McNeil [2] utilized the collection of the duodenal contents to deter-

* From the Medical Service and the Otto Baer Fund for Clinical Research of the Michael Reese Hospital, Chicago.

1. Rowntree, L. G.; Hurwitz, S. H., and Bloomfield, A. L.: An Experimental and Clinical Study of the Value of Phenoltetrachlorphthalein as a Test for Hepatic Function, Bull. Johns Hopkins Hosp. **24**: 327 (Nov.) 1913.

2. McNeil, H. L.: The Quantitative Estimation of Phenoltetrachlorphthalein Excreted in the Fresh Bile in Disease of the Liver, J. Lab. & Clin. Med. **1**: 822 (Aug.) 1916.

mine the appearance time of the dye in this fluid. Not until 1922, when Rosenthal [3] offered his modification, did the test receive any widespread use. His procedure was to inject the dye intravenously, in a dosage of 5 mg. per kilogram of body weight, and then to examine the rate of disappearance of the dye from the blood stream. Because of the difficulty often encountered in securing sharp or accurate readings, when there was present lipemia, bilirubinemia or hemolysis in the serum, Bloom and Rosenau [4] increased the accuracy of the test through acetone precipitation of the serum to be tested. Maurer and Gatewood,[5] utilizing the principle of acetone precipitation, have recently published an account of a method by which blood can be drawn from the ear by means of a blood-counting pipet, for the determination of the dye content of the serum.

Since Rosenthal presented his method, a large literature on the subject has made its appearance. Other than the developments of technic noted above, the great mass of the reports have been restricted to an evaluation of the method in the light of clinical results. The untoward reactions obtained following the use of phenoltetrachlorphthalein have been insufficiently emphasized.

UNTOWARD REACTIONS

1. *Thromboses.*—Rowntree, Hurwitz and Bloomfield, in their early work, stated that the alkali salts of phenoltetrachlorphthalein, administered subcutaneously, act as decided irritants unless they are highly diluted, as "such salts formed, as they are, by neutralizing a very weak acid with a very strong base are strongly hydrolized in aqueous solution and hence act on the tissues like solutions of caustic soda." In their clinical investigations they gave 8 c.c. of the dye, 400 mg., intravenously by the gravity method and washed through by from 50 to 100 c.c. of distilled water. Of the sixty-seven cases reported, in which this method was used, ten thromboses were encountered (over 13 per cent.). They stated that thromboses occur only when there is "marked anemia, myocardial insufficiency or advanced phlebosclerosis." Sisson,[6] using 8 c.c. of the disodium salt diluted in 100 c.c. of physiologic sodium chlorid solution, reported five thromboses in thirty-nine patients (13 per cent.). He stated that the thromboses had no relation to the disease of the patient. Chesney, Marshall and Rowntree [7] reported eight thromboses in a series of 113 cases (7 per cent.). They emphasized that thromboses frequently follow at the point of injection, as evidenced by a slight local pain or tenderness or a sense of resistance offered the palpating finger. McNeil,[2] Kahn,[8] Aaron, Beck and Schneider,[9] Williams [10] and others failed to mention the presence or absence of thromboses in their studies.

Rosenthal in his early communications gave no statistics regarding the frequency of thromboses, but advised an adequate dilution of the drug in physiologic sodium chlorid solution, as he stated that the undiluted dye might cause local irritation and occasionally thrombosis. Friedenwald and Gantt,[11] in a report covering 169 injections, commented on "several instances of thrombosis from the action of the concentrated dye on the walls of the vein at the site of injection." They reported that they had not noted a single untoward reaction, since they had been using sterile physiologic sodium chlorid solution as a diluent. In a few other communications, passing mention of the occurrence of thromboses is made.

More recently, Bloom and Rosenau [4] reported four thromboses in twenty-seven cases (14.8 per cent.). In this series, I took every precaution to prevent the occurrence of either a local reaction or a thrombosis. The dye was diluted 1 part to 2, 3 or 4 parts of sterile physiologic sodium chlorid solution and, following the injection, from 10 to 20 c.c. of physiologic sodium chlorid solution was injected, in an attempt to wash away any of the dye that might be adherent to the vein wall at the site of injection. Care was also taken to prevent any leakage of the dye into the subcutaneous tissues. These added precautions, in addition to gentle handling of the tissues, did not prevent the occurrence of local tissue reactions or thromboses. In one of the cases of thrombosis in our series, a thrombus formed in the vein from which the blood was withdrawn, as well as in the vein into which the injection of the dye had been made. The thromboses appeared from twenty-four to forty-eight hours following the injection and had the usual characteristics. Maurer and Gatewood suggest that the thromboses are perhaps due to the throwing down of the dye in colloid suspension in the blood, and the presence of the dye in this colloid form in the blood stream produces the thromboses. Rosenthal and White,[12] in their recent report advocating the use of the disodium phenoltetrabromphthaleinsulphonate test for hepatic function, state that "irritation of the vein wall is frequent, and occasionally systemic reactions occur in the use of phenoltetrachlorphthalein."

2. *Local Reactions Occurring at the Site of Injection.*—Attention has not infrequently been called to local inflammatory reactions expressed by induration, infiltration, redness, tenderness and pain at the site of injection of phenoltetrachlorphthalein. Abel and Rowntree [13] and other early workers noted the irritating action of the disodium salt, when injected subcutaneously. Rosenthal diluted the dye in physiologic sodium chlorid solution and washed the vein with the same fluid after injection, in order to avoid the local irritating action of the dye. Friedenwald and Gantt held a piece of gauze firmly around the needle when it was being withdrawn, in order to prevent any return leakage. Bloom and Rosenau reported six local reactions in twenty-seven cases (22 per cent.).

3. *Chills.*—The earliest cases of chills reported were possibly due to the use of distilled water as a diluent for the dye instead of physiologic sodium chlorid solu-

3. Rosenthal, S. M.: An Improved Method for Using Phenoltetrachlorphthalein as a Liver Function Test, J. Pharmacol & Exper. Therap. **19:** 385 (June) 1922; A New Method of Testing Liver Function with Phenoltetrachlorphthalein, J. A. M. A. **79:** 2151 (Dec. 23) 1922; A New Method of Testing Liver Function with Phenoltetrachlorphthalein, Second Communication, Bull. Johns Hopkins Hosp. **33:** 432 (Dec.) 1922.

4. Bloom, William; and Rosenau, W. H.: A Simple Method for the Determination of Phenoltetrachlorphthalein in Blood Serum, J. A. M. A. **82:** 547 (Feb. 16) 1924; The Reaction of the Liver to Phenoltetrachlorphthalein in Early Obstructive Jaundice, Arch. Int. Med. **34:** 446 (Oct.) 1924.

5. Maurer, Siegfried; and Gatewood, L. C.: Phenoltetrachlorphthalein Liver Function Test, J. A. M. A. **84:** 935 (March 28) 1925.

6. Sisson, W. R.: A Clinical Study of Two Hepatic Functional Tests (Galactose and Phenoltetrachlorphthalein), Arch. Int. Med. **14:** 804 (Dec.) 1914.

7. Chesney, A. M.; Marshall, E. K., Jr., and Rowntree, L. G.: Studies in Liver Function, J. A. M. A. **63:** 1533 (Oct. 31) 1914.

8. Kahn, Max: Phenoltetrachlorphthalein Estimation in the Duodenal Contents, J. A. M. A. **77:** 41 (July 2) 1921.

9. Aaron, A. H.; Beck, E. C., and Schneider, H. C.: Phenoltetrachlorphthalein Test for Liver Function, J. A. M. A. **77:** 1631 (Nov. 19) 1921.

10. Williams, P. F.: Phenoltetrachlorphthalein Test for Liver Function in Pregnancy, Am. J. Obst. & Gynec. **4:** 26 (July) 1922.

11. Friendenwald, J., and Gantt, W. H.: Some Observations on the Phenoltetrachlorphthalein Test as a Means of Determining Liver Function, Am. J. M. Sc. **166:** 519 (Oct.) 1923.

12. Rosenthal, S. M., and White, E. C.: Clinical Application of the Bromsulphalein Test for Hepatic Function, J. A. M. A. **84:** 1112 (April 11) 1925.

13. Abel, J. J., and Rowntree, L. G.: Pharmacologic Action of Some Phthaleins and their Derivatives, with Special Reference to their Behavior as Purgatives, J. Pharmacol. & Exper. Therap. **1:** 231 (Aug.) 1909.

tion. Sisson [6] reported a rise of temperature as high as 101 F. in one case in his series. The occasional occurrence of chills following the injection of the dye was noted by other earlier workers. In our series two chills were encountered in twenty-seven cases (7.24 per cent.). In one case the temperature rose as high as 102 F. by mouth. The chills occurred from twenty to thirty minutes after the injection. Maurer and Gatewood also reported the occurrence of chills with a rise in body temperature. They reported one case in which the temperature rose to 105 F. forty minutes following the injection. It is probable that chills have been encountered by other workers but not mentioned.

In our work we encountered ten untoward reactions (thromboses, local reactions and chills) in twenty-seven cases (37 per cent.) subjected to the phenoltetrachlorphthalein test. Because of these reactions, the number of clinical injections in the series we reported was curtailed.

4. *Deaths.*—Rowntree, Hurwitz and Bloomfield [1] reported one case of unexplained death, occurring six days after the injection of the dye. Maurer and Gatewood recently reported three patients in their series who died from two to seven days after the test was made. Two of the three cases showed recent thromboses, the dye being qualitatively demonstrated in one thrombus. These deaths may possibly have been caused by the production of fatal thromboses as a result of the injection of the dye.

5. *Other Symptoms.*—1. A sensation of dryness is a common occurrence from ten to fifteen minutes after the injection. The patients put out their tongues, lick their lips and complain of dryness of the mouth.

2. Pain over the liver region was complained of occasionally.

3. Purgative action lasting six days was reported by early workers. In our clinical series, little, if any, laxative effect was noted.

CLINICAL AND EXPERIMENTAL WORK INDICATING STRAIN ON OR DAMAGE TO THE LIVER

Early workers reported that the dye could be repeatedly injected in large quantities, over a period of many months, without any demonstrable ill effects to the animal. Bloom and Rosenau,[4] in experimental and clinical studies, called attention to a constant increase in the bilirubin content of the blood, following the injection of the disodium salt of phenoltetrachlorphthalein. Normal as well as abnormal cases showed uniformly a definite increase in the indirect van den Bergh test. When the van den Bergh test was positive by the indirect method alone, or by the direct and indirect, before the injection of the dye, there was always a uniform increase in the intensity of the van den Bergh reaction, in the direct and indirect, after the injection. The increase in the van den Bergh reaction was found at the end of one hour following the injection, and disappeared within twenty-four hours. This temporary hyperbilirubinemia seemed to bear no relation to the severity of the impairment of the liver function, as measured by the dye retention. Interpreting this hyperbilirubinemia, it was stated: "We believe that the transient bilirubinemia subsequent to the injection of phenoltetrachlorphthalein was due to the fact that the liver, having an apparently specific, excretory action toward the dye after it has been injected intravenously, cannot excrete bilirubin or its precursors which are brought to it until all of the dye has been eliminated. We have no evidence that this transient bilirubinemia is indicative of anything more than a temporary functional blockade of the bilirubin excreting power of the liver."

Because of this hyperbilirubinemia occurring constantly following the injection of the dye at that time, we felt that one should be cautious in injecting the dye in cases in which there had been a great amount of liver damage or injury. It was felt that in cases of severe liver damage, such as acute yellow atrophy of the liver, chloroform poisoning or severe cirrhosis, the injection of the dye might possibly add such a burden to the liver, in the attempt at removal from the blood stream, as to result in the subsequent death of the patient, as a result of hepatic insufficiency. A good parallel is the addition of a small strain to a badly damaged heart muscle, with a very small amount of reserve, with the death of the patient.

Recently Ottenberg and Abramson [14] have reported the production of liver necrosis in rabbits following the injection of a 5 per cent. solution of either phenoltetrachlorphthalein or tetrabromphenolphthalein in doses of 0.3 to 0.4 gm. per kilogram of body weight. Larger doses killed the rabbits at once or within fifteen minutes, with "acute convulsive symptoms." They call attention to the fact that the doses needed to produce severe symptoms and liver lesions were enormously larger than those used in the phenoltetrachlorphthalein liver function test (0.005 gm. per kilogram being equal to the clinical dosage). These observations give added significance to the observations of the occurrence of a hyperbilirubinemia, following the injection of phenoltetrachlorphthalein.

ATTEMPTS TO MAKE PHENOLTETRACHLORPHTHALEIN NONIRRITATING ON INJECTION

Phenolsulphonephthalein injected subcutaneously, intramuscularly or intravenously is followed by no local reactions or thromboses. Phenolphthalein also is nonirritating. These facts suggested that the chlorin radical might be responsible for the irritating nature of the dye. Attempts were made to neutralize a possible theoretical liberation of chlorin in the tissues, or to modify the hydrogen ion concentration of the injected dye.

The disodium salt of phenoltetrachlorphthalein was diluted in the following sterile solutions:

1. Distilled water.
2. Physiologic sodium chlorid solution.
3. Sodium bicarbonate, 5 per cent. solution.
4. Sodium carbonate, 5 per cent. solution.
5. A phosphate buffer solution.

A phosphate buffer solution, when added to the dye, decolorized it and also resulted in the formation of a white precipitate. Because of this, the buffer solution was not used in the following experiments.

Rabbits' ears were used for the injection of the dye diluted in the various solutions to determine whether or not the character of the solvent might modify the irritating nature of the dye. The dye was made up in various dilution strengths and then injected superficially in the rabbit's ear. Injection was stopped when a small wheal had been produced. A control injection of the diluent was also made. The results obtained are given in Table 1, the percentage strengths being the actual dye contents of the solutions.

From the table it can be seen that the disodium salt of phenoltetrachlorphthalein, dissolved in water, 5 per

14. Ottenberg, Reuben; and Abramson, H. A.: Production of Liver Necrosis by Tetrachlorphenolphthalein and Tetrabromphenolphthalein, J. A. M. A. 84: 800 (March 14) 1925.

cent. sodium bicarbonate, 5 per cent. sodium carbonate, or physiologic sodium chlorid solution is irritating and capable of producing inflammation on injection in the rabbit's ear, even in dilutions as low as 0.15 per cent. If one applies these results to the carrying out of the test on human beings, one would perhaps be justified in assuming that the disodium salt of phenoltetrachlorphthalein, even when diluted 1 part of dye to 30 parts of physiologic sodium chlorid solution, will possibly still be capable of producing irritation and inflammation at the site of injection. In the clinical test, a man weighing 70 kg. (150 pounds) is given 7.5 c.c. of the dye (1 c.c. containing 50 mg.). To inject 7.5 c.c. of the dye, it would be necessary to use over 200 c.c. of a diluent, to make up the 0.15 per cent. solution. The impracticability of such a procedure obviously eliminates this method from clinical use.

The untoward results described above have not been confined to the use of phenoltetrachlorphthalein. The introduction of tetrabromphenolphthalein for cholecystography has been followed by severe reaction on the part of some of the patients subjected to this test. In my own early experience with this substance, its intravenous injection into a middle aged man, for the purpose of obtaining a gallbladder shadow as a diagnostic aid, was followed within a few minutes by collapse, with a very feeble, rapid pulse. He recovered after hospitalization, but his condition following the injection was alarming.[15]

Whitaker and Milliken,[16] who used tetrabromphenolphthalein in only from 1 to 2 per cent. solution in physiologic sodium chlorid solution, asserted that they had only one reaction with headache, chills and vomiting in a series of twenty cases. Graham, Cole and Copher [17] have recently reported that a 12.5 per cent. solution of sodium tetra-iodophenolphthalein, which they use in cholecystography, at times produced induration at the site of injection. Thromboses following the injection of tetrabromphenolphthalein have also been reported by Smithies and Oleson.[18] Because of the toxicity of these substances following inravenous injection,[19] Menees and Robinson [20] have successfully administered tetrabromphenolphthalein by mouth, in capsules treated with formaldehyd to render them insoluble in the stomach, and Stewart [21] has obtained satisfactory results by the administration of tetra-iodophenolphthalein into the jejunum through a duodenal tube.[22] It has been concluded that "in spite of the toxic properties of the drug no fatal results due to its administration have been reported, but there is no doubt that much harm and even fatalities might be caused by overdosage." [19]

It would therefore appear that the bromin and iodin phthalein compounds used in cholecystography possess to greater or lesser degree the irritating and toxic properties of phenoltetrachlorphthalein. I cannot help but advise against the indiscriminate injection of these halogenated phthaleins, especially because I feel that additional chemical research and further animal experimentation will probably result in the introduction of compounds that are as innocuous as phenolsulphonephthalein has proved to be and as Rosenthal and White [12] claim disodium phenoltetrabromphthaleinsulphonate to be. They recommend the use of disodium phenoltetrabromphthaleinsulphonate as superior to phenoltetrachlorphthalein for liver function work, in that it is a more delicate test and is nonirritating. In fifty tests, on human beings, no untoward effects of any kind were experienced.

TABLE 1.—*Reaction Observed in the Ears of Rabbits Twenty-four Hours After Injection of Phenoltetrachlorphthalein Dissolved in Various Solutions*

Diluents	Diluent Unchanged 5%*	2.5% Sol. Dye	1.2% Sol. Dye	0.6% Sol. Dye	0.3% Sol. Dye	0.15% Sol. Dye
Distilled water.....	No reaction	Necrotic ulcer	Slight ulceration	Well marked infiltration	Infiltration	Slight infiltration
5% sodium bicarbonate	No reaction	Necrotic ulcer	Slight ulceration	Well marked infiltration	Infiltration	Slight infiltration
5% sodium carbonate	No reaction	Necrotic ulcer	Slight ulceration	Well marked infiltration	Infiltration	Slight infiltration
Physiologic sodium chlorid solution	No reaction	Necrotic ulcer	Slight ulceration	Well marked infiltration	Infiltration	Slight infiltration

* Five per cent. solution of disodium salt of phenoltetrachlorphthalein contained in ampules put out by Hynson, Wescott and Dunning.

SUMMARY

Following the use of phenoltetrachlorphthalein, thromboses, local inflammatory reactions at the site of injection and chills have been encountered by many observers. Several deaths probably due to its use have been reported.

Clinical and experimental work indicates the possibility of strain or damage to the liver, following injection of the dye.

Attempts to make phenoltetrachlorphthalein nonirritating on injection have failed.

Many observers have noticed severe toxic reactions following the use of tetrabromphenolphthalein and tetra-iodophenolpthphthalein.

The toxicity of the halogenated phthalein compounds should lead to caution in their use. Indiscriminate administration and overdosage should be avoided.

15. Dr. J. Friedman has kindly given me permission to report this case.

16. Whitaker, L. R., and Milliken, G.: A Comparison of Sodium Tetrabromphenolphthalein and Sodium Tetraiodophenolphthalein in Gallbladder Radiography, Surg., Gynec. & Obst. **40**: 17 (Jan.) 1925.

17. Graham, E. A.; Cole, W. H., and Copher, G. H.: Cholecystography, the Use of Sodium Tetra-Iodophenolphthalein, J. A. M. A. **84**: 1175 (April 18) 1925.

18. Smithies, Frank; and Oleson, R. B.: Aseptic Irritative Phlebitis, Following Intravenous Injection of Sodium Salt of Tetrabromphenolphthalein, J. A. M. A. **84**: 1494 (May 16) 1925.

19. The Limits of Safety in Administration of Tetrabromphenolphthalein, editorial, Am. J. Roentgenol. **13**: 371 (April) 1925.

20. Menees, T. O., and Robinson, H. C.: Oral Administration of Sodium Tetrabromphenolphthalein: Preliminary Report, Am. J. Roentgenol. **13**: 368 (April) 1925.

21. Stewart, W. H.: Administration of Tetra-Iodophenolphthalein through Duodenal Tube, Am. J. Roentgenol. **13**: 378 (April) 1925.

22. Since this article was prepared, two reports of the successful oral administration of sodium tetra-iodophenolphthalein in cholecystography have appeared: Einhorn, Max; Stewart, W. H., and Ryan, E. J.: Experiences with Cholecystography, M. J. & Rec. **122**: 77 (July 15) 1925. Graham, E. A.; Cole, W. H.; Moore, Sherwood; and Copher, G. H.: Cholecystography: Oral Administration of Sodium Tetra-Iodophenolphthalein, J. A. M. A. **85**: 953 (Sept. 26) 1925. Satisfactory gallbladder pictures were obtained, and this method of oral administration was found to be relatively nontoxic.

Insanity and Marriage.—When there is evidence of insanity or epilepsy in both stocks, or a general neuropathic tendency indicative of mental instability, the liability to dementia praecox is greatly increased in the descendants; therefore in advising (as one often has to do) in regard to marriage, evidence pointing to these special forms of mental disease in both stocks, on one side, still more on both sides even if shown in collaterals, induces one to say definitely from a eugenic point of view that it would be inadvisable—inasmuch as the risk of an insane inheritance for the children is so much the greater.—Mott, Frederick: The Harveian Oration on Some Developments of Harvey's Doctrine, "Omne vivum ex ovo," *Lancet*, Oct. 24, 1925.

THE GALLBLADDER AS REVEALED BY THE ROENTGEN RAY

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More than twenty years ago when Rieder gave the first bismuth meal, it was observed that the stomach was not located horizontally in the epigastrium, but that, in the average person, it hung vertically on the left side of the abdomen, its lower pole reaching to the level of the navel and usually below the navel. This observation was not immediately accepted by clinicians, because it did not seem to agree with the observations made, over a long period of years, by surgeons doing abdominal operations and by pathologists while making necropsies. The unique value and accuracy of roentgen-ray stomach visualization is now universally accepted, however.

The visualization of the gallbladder by the use of the tetra-iodophenolphthalein test, as brought out by Graham and his co-workers, promises to be an equally important milestone for clinical medicine. The oral administration now permits of universal application of this test without danger or discomfort to the patient.

During the last four months we have used the tetra-iodophenolphthalein test by the oral method to visualize the gallbladder in 500 routine abdominal roentgen-ray examinations. By perfecting the technic, we have been able to obtain, in the last 200 cases, a distinct gallbladder outline in more than 90 per cent. of the normal cases. A comparative study of the films obtained in this series shows that the normal gallbladder varies so greatly in location that we are impelled to ask: "Where is the normal gallbladder?" In answer thereto, we feel justified in saying that in the great majority of persons the gallbladder is not in the gallbladder region as clinically understood. This rather bizarre statement will demand some further explanation and discussion.

The gallbladder region, as given in Morris' Anatomy, is the right hypochondrium and epigastrium. The longitudinal line that separates the right hypochondrium from the epigastrium is said to cut the gallbladder. The lower limit of the right hypochondrium and epigastrium is the line connecting the tenth costal cartilages, and this line bisects the body of the second lumbar vertebra.

In my series of cases, the gallbladder extends either partly or wholly below the lower limits of the hypochondriac and epigastric regions in 75 per cent. of the cases. Usually it descends into the umbilical region but quite frequently into the right lumbar region.

Furthermore, in the 25 per cent. of the cases in which the gallbladder is found above the second lumbar vertebra, that is, in the so-called gallbladder region, it does not always lie in the line that separates the hypochondrium and epigrastrium, but may lie far to the right of this line and may be found outward as far as the axillary margin of the abdomen. The clinical importance of this observation can be readily understood. I have recently observed a case of intermittent hydrops of the gallbladder in which the patient complained of pain in the axillary margin of the hypochondrium (that is, between the hepatic flexure of the colon and the right kidney), and the dye test in this case revealed a distended gallbladder, the fundus of which approached the axillary margin of the chest. In another instance, a palpable and freely movable tumor mass was found in the axillary margin of the right hypochondrium, approaching the right kidney region. Operation revealed a hydrops of the gallbladder. Such a lateral lying gallbladder may cause pain, tenderness or a tumor mass simulating a loose kidney, kidney tumor, tumor of the hepatic flexure or disease about an ascending retrocecal appendix.

Of those gallbladders found below the right hypochondrium and epigastrium, that is, below the level of the second lumbar vertebra, some extended only to the level of the third lumbar vertebra, but a surprisingly great number were found with the fundus approaching the fourth lumbar vertebra or level of the umbilicus and crest of the ilium, and some extended into the lumbosacral angle and to the level of the fifth lumbar vertebra. In most instances the fundus of the gallbladder seemed to point toward the midline of the body and the navel, but in a few instances the fundus was directed outward, approximating the anterior superior spine of the ilium. In one case I found a gallstone lying just above the anterior superior spine. Before giving the dye I had overlooked this stone, as I interpreted it as fecal material in the ascending colon, but the administration of the dye showed the location of the gallbladder and led to a correct diagnosis.

A very striking observation is the fact that the low gallbladders frequently cast their shadows very close to the spine and in some instances overlap the spine. Certainly, many gallstones have been overlooked in the past, being obscured by the shadows of the spine. This brings us to a very important point in technic. In all the cases included in this series, the roentgen-ray examination was made with the patient lying prone, and the tube was centered vertically over the gallbladder, which would avoid any upward or downward (cephalad or caudad) projection of the gallbladder shadow. However, in order to avoid superimposition of the shadow of the spine on the shadow of the gallbladder, it was necessary in many instances to rotate the body slightly to the left. In spite of this position, however, we still obtained some gallbladder shadows partly obscured by the shadow of the spine, which would prove that a large percentage of gallstone shadows have been obscured in this way.

To determine the range of mobility of the gallbladder, films were made also in the supine and erect postures. A comparison with the routine films made prone showed that the gallbladder moves upward from one-half to 1 inch, when the patient changes from the prone to the supine position. When the patient assumes the erect posture, the gallbladder descends from 1 inch to 1½ inches. This excursion of the gallbladder is least in well nourished or stout persons having good muscular tone. In the thin, relaxed persons, the difference between the erect and the recumbent postures is often surprisingly great, sometimes as much as 3 or 4 inches. Of course, a certain part of the excursion of the gallbladder is due to the movement of the liver and diaphragm, which always accompanies changes in posture.

A diseased gallbladder whose fundus reaches to or extends below the navel may clinically simulate a diseased appendix. Routine roentgen-ray examinations of the colon have shown us that the appendix is usually much below McBurney's point and in most instances is seen deep in the pelvis, but a diseased appendix may refer its pain to the umbilical region, which will explain the frequent difficulty in differentiating gallbladder and appendix disease.

A very low gallbladder, if diseased, may even simulate disease of the right ovary.

The fact that gallstones may be found below the right hypochondrium and epigastrium is not a new observation. Carman, in his book on "The Roentgen Diagnosis of Diseases of the Alimentary Tract," which was written before the introduction of the roentgen-ray visualization of the gallbladder, states that gallstones may be found as low as the crest of the right ilium, and that the entire region from the ribs to the ilium should be covered. However, I do not believe that the great variation of the position of the gallbladder has been sufficiently emphasized, as it was formerly not possible to obtain and to study the gallbladder outline under normal conditions.

Students have been taught to palpate the gallbladder high under the ribs. Roentgen-ray operators, in making fluoroscopic palpations of the abdomen, usually palpate too high for the gallbladder. Fluoroscopic palpations are very valuable diagnostic procedures, especially if the colon is filled by a previous barium meal. The location of the transverse colon, whether high or low, will be the dividing line between the gallbladder region and the appendix region. The hepatic flexure is not a safe guide, as the gallbladder will frequently descend along the mesial aspect of the hepatic flexure to come in contact with a sagging transverse colon.

The variations in the position of the gallbladder are of importance chiefly to the clinician. The surgeon has his landmarks to guide him and will readily find the gallbladder irrespective of its location. The surgeon states that he usually is obliged to pull the gallbladder from under the liver; but at operation the patient is supine and (except when local anesthesia is used) is unconscious. It is, furthermore, likely that preparation for operation, nervous excitement, fasting, struggling or some action of the anesthetic may explain the small, high, contracted gallbladder so frequently observed at operation. It is interesting to note in this connection that before the introduction of the bismuth meal, no surgeon had noted or commented on the position of the normal stomach, as we now understand it.

If the tetra-iodophenolphthalein method of gallbladder visualization does nothing more than to give us the size, shape and position of the gallbladder, it will serve an important purpose in gallbladder diagnoses; but this method has a much wider clinical application.

We are now employing this method as a routine in every complete abdominal examination and are obtaining gallbladder outlines in 90 per cent. of the normal cases. From the standpoint of exclusion (and many roentgen-ray abdominal diagnoses are made partly by exclusion), the clinical value and importance of being able to see the location, size and shape of the gallbladder can hardly be overestimated. In many cases (although not in all) the location of the patient's pain bears a direct relation to the location of his lesion, and the location of the gallbladder with reference to the location of the pain can thus be determined. In another group of cases, a tender point in the abdomen will correspond with the location of the lesion, and the position of the gallbladder with reference to the tender point can be studied. In another group of cases, there may be a palpable abdominal tumor present, and again this method will enable us to determine the relation of the gallbladder to the location of the tumor mass.

It frequently happens that a shadow appears on the roentgenogram that cannot be identified. By obtaining an outline of the gallbladder, we can note whether this shadow corresponds with the position of the gallbladder. If the suspicious shadow corresponds with the shadow of the gallbladder, oblique and lateral views can be made in order to prove whether the shadow falls within the gallbladder outline or whether in front of or behind it. If the suspicious shadow does not correspond in location with the gallbladder shadow, gallstone is immediately and positively excluded.

Since the dye does not enter the gallbladder freely in the presence of a lesion of the gallbladder or bile ducts, the clinical deductions above mentioned may not always be possible. Frequently we obtain only a very faint gallbladder outline which must be carefully searched for, and such a faint outline is always suggestive of a gallbladder lesion. A total absence of the gallbladder outline will prevent the carrying out of the foregoing tests, but the absence of the gallbladder shadow points, in itself, to the gallbladder or liver as the possible site of the lesion.

In those cases in which we have obtained no shadows in the gallbladder region suspicious of gallstones and in which the tetra-iodophenolphthalein test has given a very distinct outline of the gallbladder, and which appears of average size (that is, not unduly distended or contracted) and which does not appear deformed or irregular, we have felt safe in reporting a normal gallbladder. Great distention of the gallbladder would indicate a hydrops. Deformity of the outline would indicate inflammatory changes in the walls of the gallbladder or possibly adhesions about the gallbladder. We have recently seen several cases in which it was horizontally placed and badly deformed, and in which operation revealed a diseased gallbladder adherent to the under surface of the liver. A very interesting condition is that in which the gallbladder outline shows only faintly and is not of uniform density, showing one or more dark areas. These dark areas have been proved to be transparent gallstones which prevent the dye from completely filling out the gallbladder.

When the gallbladder shows only faintly or fails to show at all, our suspicion is directed to it, although we cannot immediately make a diagnosis of a gallbladder lesion without further study. Advanced disease of the liver, such as cirrhosis (which would prevent the normal secretion of the bile) must be first excluded. Then, the failure of the gallbladder to fill with the dye may be due to obstruction of the bile passages by a stone, by thickened mucus or by swollen or edematous walls. The presence of inspissated mucus or bile in the gallbladder would prevent the dye from entering. When the gallbladder fails to fill with the dye, the test should be repeated in order to check up the findings and in special cases, when the clinical aspects of the case do not seem to confirm the diagnosis of a gallbladder lesion, the intravenous injection of the dye may be resorted to. If diseases of the other abdominal organs have been excluded, as far as is possible by the roentgen ray and by a thorough clinical examination, the failure of the gallbladder to visualize, would justify a diagnosis of a gallbladder lesion, especially if the patient presents a confirmatory clinical history. A close coöperation between the clinician and the roentgenologist is essential for a correct diagnosis, as is true of all roentgen-ray work.

The fact that in approximately 10 per cent. of the normal cases the gallbladder may fail to show would lead to a possible error of 10 per cent. Repetition of the test in doubtful cases may, however, reduce this percentage of error. It is furthermore possible, that some of the gallbladders which visualize and are reported normal may be found to be diseased, which

fact may increase the chance of error. Much clinical study and further statistical observation are necessary before the final diagnostic accuracy of this method can be definitely stated.

Of those cases in my own series which were reported normal and which later came to operation, no errors have been noted up to this time, although only a small percentage have been checked by operation. Of those cases in which the gallbladder failed to visualize and a gallbladder lesion was reported, only one error has been noted, although again the percentage of cases in which operation has been performed has not been very great. In all of the operative cases in which gallstones were reported, gallstones have been found.

One strikingly uniform observation in my series of cases, which would seem to prove the basic rule that abnormal or diseased gallbladders do not fill with the dye as completely and uniformly as do the normal gallbladders, is the fact that in those cases in which a preliminary roentgenogram has shown the presence of gallstones, the subsequent dye test would either show a failure of the gallbladder to fill or would reveal a faint and incompletely visualized gallbladder. When the gallstone blocks the cystic duct, the failure of the gallbladder to fill may be purely mechanical, but in the many gallstone cases in which the ducts are not blocked, the failure of the gallbladder to show in the usual way must be attributed to associated disease of the gallbladder or bile passages.

Further evidence on this point has been obtained from a study of the results of the dye test when made on slightly or deeply jaundiced patients. It has been our uniform finding that the gallbladder will fail to visualize in the presence of jaundice, irrespective of whether the jaundice is obstructive or infectious. In this connection it may be well to bring out the fact that no untoward effects have been observed following the administration of the dye to jaundiced patients.

Prior to the introduction of the tetra-iodophenolphthalein test, roentgenologists have attempted to diagnose gallbladder disease by obtaining a faint outline of the gallbladder. When the gallbladder outline was visible, a thickened or distended gallbladder was reported. In order to test the accuracy of this method, we have attempted to identify the gallbladder shadow on the preliminary film, then checking the finding after administering the dye. In the majority of instances, the preliminary identification of the gallbladder shadow was incorrect, and often the gallbladder was located some distance from the suspected shadow. It is therefore apparent that many inaccuracies have in the past attended such a method of diagnosis. The use of a small cone (as was formerly recommended) would, for obvious reasons, increase the chance of error.

In every case in which the gallbladder fails to visualize, it is of prime importance to make an immediate fluoroscopic examination of the abdomen to search for undissolved capsules. Irrespective of the type of coating used, it frequently happens that the capsules will fail to dissolve. If the majority of the capsules are undissolved, the test should be repeated a second or even a third time. We have thus been able to visualize gallbladders in certain cases in which a cholecystitis had been erroneously diagnosed on the basis of a supposedly absent gallbladder shadow.

Irrespective of the thickness of the abdomen, all roentgenograms of the dye-filled gallbladder have shown an unusually distinct outline of the right kidney, due possibly to the presence of a small amount of the dye in the kidney. The relations of the gallbladder shadow to that of the right kidney have been so varied as to deserve comment. The gallbladder shadow may be well above the kidney shadow, may lie directly in front of the renal pelvis, or may be mesial or external to the kidney outline. Not infrequently, however, the gallbladder shadow has been found to be well below the renal pelvis or even below the lower pole of the kidney. Gallstone shadows may, therefore, be much lower than shadows of renal calculi.

Certain patients in this series have presented, as their chief complaint, pain in the right hypochondrium or directly under the right rib margin, suggesting gallbladder disease. In a certain percentage of such cases an apparently normal gallbladder outline has been found at or near the level of the navel and quite a distance below the site of the pain. In many of these cases the completed roentgen-ray examination has revealed nothing abnormal except a general visceroptosis. The pain in the hypochondrium must, therefore, be attributed to traction of the prolapsed viscera. The dye test will thus serve to exclude gallbladder disease in this very prevalent type of case.

Doctors Building.

MIXED CELL TUMOR OF THE PALATE

REPORT OF CASE *

G. V. BOYKO, D.D.S.
PASSAIC, N. J.

Tumors of the palate, composed in part of epithelial-like cells, with noteworthy hyaline and mucoid degeneration of both these cells and the connective tissue framework, frequently have been classified as endotheliomas, especially by the earlier writers. The tendency now is to consider them as arising from the embryonic clefts. It is also generally believed that they consist of epithelial cells with varying proportions of connective tissue, cartilage and other tissues characteristic of this region. They are for the most part benign, since as a rule they do not infiltrate, are encapsulated and do not produce metastases.

REPORT OF CASE

History.—A woman, aged 32, came to the surgery clinic of Northwestern University Dental School, Feb. 13, 1925, for advice concerning a swelling of the palate of four years' duration. At first no larger than a small marble, it gradually increased in size, but at no time did it give rise to actual pain or soreness. From a place to the right of the midline and opposite the molar region, it spread in all directions until it occupied most of the space between the molar teeth. About two years before she was admitted, the patient consulted a physician, who lanced this swelling, producing a flow of blood that lasted for several minutes. Since that time, the mass had increased in size, as before, but it was not until three or four months before we saw her that the tumor, because of its size, became embarassing.

Examination.—A tumor mass, with a smooth covering of mucous membrane and about the size of a large walnut, extended from the gingival border of the right molars to within one-fourth inch (6 mm.) of the left molars, and from the posterior extremity of the hard palate to within 1 inch (25 mm.) of the gingival border of the incisors. There was an overlapping of the sides of the tumor against the teeth and the tissues of the palate and it was neither painful nor tender. There was no evidence of any metastasis, and the patient was otherwise in good health.

* Read before the Section on Stomatology at the Seventy-Sixth Annual Session of the American Medical Association, Atlantic City, N. J., May, 1925.

Operation.—The patient was admitted to St. Luke's Hospital, Chicago, February 26, and the tumor was shelled out *en masse* on the following day under ether spray, general anesthesia by Dr. H. A. Potts. It was necessary to remove a considerable portion of the mucous membrane of the palate with the tumor; the site of the tumor was allowed to fill in with granulation tissue over which epithelium gradually spread. The tumor mass was enclosed by a fibrous tissue capsule and was attached to the palate by a flat, wide pedicle to the right of the median line. At this place, there was noteworthy absorption of the bone of the palate; in the center of this region, the bone was entirely missing and this defect was 3 mm. in diameter.

Microscopic Examination.—Sections of this tumor stained with hematoxylin and eosin, hematoxylin and van Gieson's fluid, contained nodules and cords of round and oval epithelial cells in an abundant connective tissue framework. In places, the intercellular substance of the connective tissue matrix was so abundant as to form distinct bands. There were many regions of mucoid degeneration which took the blue stain in a diffuse manner. In such regions, the epithelial cells were swollen and the nuclei were crowded toward the periphery of the cell. In other places, these cells had an arrangement suggestive of gland forms. No mitotic figures were seen. Blood vessels were abundant and comparatively large.

SUMMARY

Although this tumor did not contain the variety of tissues found in the mixed tumors of the mouth, it seemed to belong to this group. Its unilateral origin, the tendency of the cells to undergo hyaline and mucoid degeneration and the arrangement of the epithelial cells in cords, nodules and even glandlike aggregations all are in favor of such an identity. Such tumors, while primarily benign, occasionally become malignant; on this account alone they should be removed as early as possible.

ABSTRACT OF DISCUSSION

DR. GEORGE M. DORRANCE, Philadelphia: If we study the normal roof of the palate microscopically, we shall notice that there is a large area of fibrous tissue surrounding the little glands in the roof of the mouth. It is quite easy to conceive that, as in the other type of tissue elsewhere in the body, this is glandular tissue surrounded by fibrous tissue. These tumors have not been satisfactorily classified. We used to call them fibromas. I do not think that tumors can be classified to suit all of us. It is hard to be dogmatic and say that it is a fibroma, an adenoma or a myoma. We are pretty sure when it is a carcinoma, but we are not sure of the other tumors. The treatment of this case is ideal. The tumor was taken out entirely, and more than likely there will be no recurrence. At the Philadelphia General Hospital, where we have a group of seven men, including a roentgenologist and a pathologist, we are continually having discussions as to where to classify these tumors. Until such time as the pathologists and the clinicians agree, we shall have to classify them with the old group of fibromas and adenomas. However, I find that these tumors are very rare. The ordinary osteoma is common. The results of this seem to me to follow along with hypertrophy of the gums. We do not know what causes hypertrophy of the gums. We know when it occurs, we remove the tumors completely and they do not return. The same is true of this tumor.

DR. ROBERT H. IVY, Philadelphia: My experience has been limited to two tumors of this type, one confined to the soft palate and the other to the hard palate. Both of them exhibited microscopic characteristics identical with this one of Dr. Boyko's. The sections from the first tumor, which occurred about twelve years ago, were submitted to practically all the leading pathologists in Philadelphia, and there was great disagreement as to what it was. Several of them felt that it was endotheliomatous in nature, and it was as such that I reported the tumor, but I feel convinced now that these cells are epithelial and that we must not classify them as endotheliomas, in which group a great many tumors have been put when they could not be definitely assigned elsewhere.

DR. F. A. FIGI, Rochester, Minn.: I hardly see how this tumor can be classified as either an adenoma or a fibroma. The possibility of considering it a fibroma is ruled out by the marked preponderance of epithelial elements present, while the atypical character of these cells precludes its being an adenoma. I would say rather that it was a mixed tumor or an adenocarcinoma. Both of these frequently occur in this region, and both are comparatively benign types of malignant tumor. They are both definitely malignant, however. They may occur either in the hard or in the soft palate and often reach an enormous size. We have removed them from this region as large as 8 or 9 cm. in diameter, after ligation of the external carotid artery on either side. I think this case was handled ideally from the standpoint of treatment.

DR. EDWARD H. HATTON, Chicago: The fact should not be lost sight of that the structures of the palate and the parotid region of the neck, and to a less degree of the lips and the tongue, are intimately connected with the embryonic gill clefts, and that in the closing of these gill clefts abundant opportunity is offered for the deposition of cell rests containing rudimentary epithelium, cartilage, fibrous tissue and sometimes glands. Tumors arising from these rests are unilateral, slow growing, stimulated to growth at the time of puberty or afterward, and develop much as this one did. On this account I am inclined to believe that the mixed cell classification is correct and that these cells are epithelial cells.

DR. CARL W. WALDRON, Minneapolis: My experience has been limited to two cases, both of which were somewhat interesting. In one case, the mixed tumor of the palate was no larger than the diameter of a fountain pen it was situated in the center of the hard palate at about the junction of the maxillary and palatal bones. It had developed slowly over a period of three or four years at the site of an injury made by a lead pencil some fifteen years before. It was very readily removed without recurrence. The other case was a somewhat unusual one. A patient came to our clinic with very marked protrusion of the upper six anterior teeth, so marked that the upper lip was forced upward beneath the nose, and the lip could not be closed, even on strongest muscular effort. The whole anterior portion of the alveolar process with the teeth was resected *en masse*, and a very favorable result from the standpoint of a denture was secured. About a year later she returned, complaining that the denture did not feel comfortable posteriorly on the palatal side of the tuberosity. At this site she had been operated on twice previously and had been told that there was dead bone. Fortunately, at the first appearance of the patient, we took a cast of this unusual deformity, which showed a slight asymmetry in this palatal region. On reexamination, this asymmetry was more pronounced. Palpation seemed to show very definite fluctuation, and on incision some material came away, not a liquid exudate or purulence, but rather like softened lymphoid tissue, such as one might encounter on incising a tonsil capsule after its removal in a broken down tonsil. I collected this for examination, and the report was a mixed tumor. I operated again a week or two later, but in this instance the swelling was so small that to the best of my ability I could not determine a capsule, so I excised, I felt, sufficiently widely to remove the tumor. However, it may be subject to recurrence, and I shall check it up as soon as I return.

DR. G. V. BOYKO, Passaic, N. J.: As Dr. Hatton has brought out in his discussion, the characteristic structure of this tumor and the fact that there is no definite classification for it make it seem most appropriate to place it in the "mixed tumor" class.

Smallpox in Connecticut.—During the years 1923 and 1924, 152 cases of smallpox with six deaths were reported in Connecticut. Five of these deaths occurred in persons who had never been successfully vaccinated and one had been vaccinated more than seven years before onset. In the first group mentioned there were ninety-six cases; in the second group, twenty-four cases. One patient had had smallpox previously, and three patients had been vaccinated within seven years.—*Pub. Health Rep.* **40**:1811 (Aug. 28) 1925.

NUTRITIONAL KERATOMALACIA

REPORT OF CASE

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MILWAUKEE

The occurrence of keratomalacia as a result of faulty diet has been established for human beings as well as for animals.[1] While numerous observers realized early that the disease had something to do with nutrition, and many conceived a definite relationship to the diet, it was not until the striking animal experiments of McCollum and his co-workers,[2] and those of Osborne and Mendel,[3] that this disease could be classified as an avitaminosis. It is now generally conceded that it is essentially a deficiency disease following deprivation of the fat soluble A vitamin. Groups of cases as have been described have generally occurred in periods of famine or in countries having grossly inadequate food resources. The large series of cases collected by the Danish Ophthalmological Society during the period from 1909 to 1920 [4] were attributed to the peculiar circumstance of a dairy country exporting the most important products providing the fat soluble A factor.

McCollum's [5] recent experiments indicate that other factors besides the amount of fat soluble A contribute to the production of this disease. The total amount of salt mixture and its composition is an important factor. The interrelationship between certain nutritive principles in the dietary may play a rôle. These experiments may explain the occurrence of keratomalacia in infants who have not been completely deprived of fat soluble A. Thus, the patients reported by Ross [6] had had previous diets of condensed milk, a food not entirely lacking in this specific vitamin.

A study of the literature reveals numerous similar instances. Very frequently the history of previous digestive disturbance is reported. That functional disturbance of the digestive tract produces changes in the intermediary metabolism is well recognized. Meyer [7] found a negative salt balance in infants suffering from dyspepsia and receiving a low fat diet.

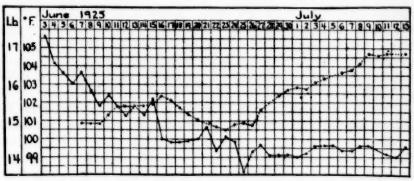
Some students of the disease distinguish several types, depending on whether the vitamin A deficiency has been the sole cause of the disease or whether other factors are also involved.

Bloch [8] identifies three types: (*a*) xerophthalmia with carbohydrate dystrophy; (*b*) xerophthalmia with carbohydrate atrophy, and (*c*) a group in which he realized that the symptoms were due to complete absence of fat substances which he termed dystrophia alipogenetica. He was then unacquainted with McCollum's work. The first two groups gave nutritional pictures corresponding to the "mehlnährschaden" of Czerny, and Bloch entertained the possibility of this being an avitaminosis in a class by itself.

Stransky [9] divided keratomalacia into two classes, one in which there is a definite avitaminosis caused by the entire absence of fat soluble A vitamin and which corresponds to experimental keratomalacia. He regards the term hypovitaminosis as more suitable for those cases in which the body's available supply has been disturbed by infection or by quantitative inanition.

In this country the universal availability of milk as a basis for infant feeding probably accounts for the rare incidence of the disease. It is interesting to note that in the years from 1890 to 1894, Kollock [10] of South Carolina observed numerous instances of the disease among the negroes. These observations formed the basis of several reports to the American Ophthalmological Society. Kollock noted that it was more common among the blacks than the mulattoes. He recognized that dietary factors played a part, noting that "the negro lived commonly on hominy (grits), rice, fish and bad hog meat," and thought that "the diet may have something to do with the condition." Kollock [11] reports that the disease is rarely observed at the present time.

Besides Kollock's observation, there have been isolated clinical reports in American medical literature by Weeks,[12] in 1887, Evans,[13] in 1898, and Little,[14] in 1882. More recently, Ross [6] reported four cases from the Children's Department of the Johns Hopkins Hospital, and DuBois and Wilson [15] reported one case from the Boston Infants' Hospital. All of these patients died from the disease.



Temperature and weight curve, showing period of three weeks' treatment before temperature (solid line) reached normal or weight (dotted line) increased.

REPORT OF CASE

The present report is one of keratomalacia following a diet therapeutically restricted, constituting virtually an experimental production of the disease.

A. J., an infant boy, had been breast fed until 10 months of age. Occasionally, besides breast milk, he received toast or buttered bread. Between the sixth and eighth months, during the past winter, he had had two bowel upsets, both occurring during a month's interval. These were cured by restricting his feedings.

The present illness began, May 12, 1925, with fever, restlessness, constipation and a slight cough. On the third day of the illness, he was reported as having had a "toxic dyspnea" and "looked as though he were going to die." This condition cleared up in thirty-six hours. After a few days his condition

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3. Osborne, T. B., and Mendel, L. B.: Ophthalmia and Diet, J. A. M. A. **76:** 905 (April 2) 1921.
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11. Kollock, C. W.: Personal communication to the author.
12. Weeks, J. E.: Xerosis Conjunctivae bei Säuglingen und Kindern, Arch. f. Augenh. **17:** 193, 1887.
13. Evans, T. C.: Keratomalacia, Arch. Pediat. **15:** 756, 1898.
14. Little, W. S.: Kerato-Malacie (Sphacelus) with a Case, Philadelphia M. Times **12:** 490, 1882.
15. Wilson, J. R., and DuBois, R. O.: Report of a Fatal Case of Keratomalacia in an Infant, with Postmortem Examination, Am. J. Dis. Child. **26:** 431 (Nov.) 1923.

became worse again. He had no more dyspnea, but had very intractable constipation, which took castor oil and frequent flushings to modify. Besides the constipation, the only other symptom that impressed the parents was the infant's stupor.

The diet instituted on the first day of illness was oatmeal water and Mellin's Food in water. Both of these were given in small quantities about every hour or two. This constituted the entire diet regimen from May 12 to June 3, a period of twenty-two days, after which the baby was admitted to the Milwaukee Infants' Hospital.

On admission, the infant presented the semistuporous appearance of a typhoid or miliary tuberculosis, not the acutely ill facies of an intestinal intoxication. The eyes were held closed most of the time. The skin was doughy, the musculature flabby and the approximate weight loss since the onset of illness about 6 pounds (2.7 kg.). The mouth was dry and congested and the lips were cracked. No generalized adenopathy was found. The heart and lungs were apparently normal, the abdomen was distended and the visceral pattern very distinct. The throat was reddened. The ear drums were normal. The liver edge seemed very sharp and firm; the spleen was not felt. There was no rigidity of the extremities or of the neck. The reflexes were not abnormal. The fingers appeared slightly cyanosed. The temperature, on admission (4 p. m.), was 105.6. The provisional diagnoses were: (1) erythredema; (2) typhoid fever, and (3) miliary tuberculosis.

The urine showed a trace of albumin, and the microscopic examination was negative. Blood examination showed: 4,800,000 red blood cells; 85 per cent. hemoglobin (Tallqvist); 13,500 leukocytes; 78 per cent. polymorphonuclears, and 22 per cent. large lymphocytes. The Pirquet reaction was negative. Intracutaneous injection of tuberculin (0.01 mg.) was negative. The Widal test was negative. Spinal fluid examination revealed: no increased pressure; clear fluid; no cells; globulin negative and the sugar reduction diminished.

During the first twenty-four hours of his hospital stay the infant was given about 8 ounces (236 c.c.) of fat free lactic acid milk containing two-thirds ounce (20 c.c.) of Karo Corn Syrup. He seemed rather eager for this feeding but vomited a large part of it. Toward the end of the twenty-four hour period, he began to retain more of his food.

June 4, the temperature ranged from 101 to 104, and the baby was in a drowsy stupor. The legs were held in a frog-like position, and the baby cried when they were touched or lifted. On the abdominal wall, to the left of the umbilicus, were several pin point petechial spots, probably the result of scratching. The possibility of scurvy suggested itself. The spots cleared up after a few days. Both eyes were dull in appearance, the sclerae yellow tinged, the right eye showing a corneal ulcer.

June 5, the child was in a stupor all the time, occasionally giving a sudden cerebral cry, accompanied by fixation of the eyes and rigidity of the upper extremities lasting only a few seconds—a miniature convulsion. The breathing was regular and quiet. There was a very slight suggestion of neck rigidity, and no rigidity of the extremities. The abdomen was distended. The cyanosis of the hands had disappeared. Spinal fluid examination was negative. The corneal ulcer was more distinct.

A diagnosis of keratomalacia being made, the food was changed to whole lactic acid milk, and 10 drops of cod liver oil three times daily was ordered. One ounce (30 c.c.) of orange juice was given twice daily.

Eye examination by Dr. Haessler revealed a large loss of substance in the cornea of the right eye, 4 mm. in diameter, involving one half of the depth of the cornea, with almost no marginal infiltration. Minimal hyperemia of the conjunctiva and no demonstrable ciliary hyperemia were discovered. The remainder of the cornea was clear, though the epithelial surface was slightly dull and distinctly less sensitive to mechanical stimuli than normal. The pupil did not dilate under homatropin, but the corneal defect would preclude ophthalmoscopic examination.

The corneal surface of the left eye was dull and less sensitive than normal and seemed slightly dry and stippled. A very faint nebula was seen in stroma by oblique illumination and was more evident by transmitted light. The fundus seen through a 5 mm. pupil (homatropin) was essentially normal. A slight haze of nerve head margin was ascribed to the effect of corneal imperfection. The diagnosis was trophic corneal lesion (xerophthalmia).

June 6, the patient was less drowsy, but kept his eyes closed and fretted while being examined. He cried out occasionally as though in pain. He took his feedings willingly. Though the tongue looked parched and dry, the mouth condition was slightly improved. The abdominal distention was less pronounced and the tissue turgor poor.

June 7, the child moved his hands to his mouth constantly, and seemed much fussier and more fretful when his legs were moved, the left one particularly distressing him. Examination of the right eye revealed the ulcer edge to be deeply infiltrated with heavy white margin, and the hyperemia markedly increased.

Cod liver oil, 1 c.c. hypodermically, was given daily.

June 8, the ulcer edge of the right eye was not greatly changed. As the center of the ulcer bulged moderately, a pressure bandage was applied.

Blood examination revealed: white blood cells, 11,500; polymorphonuclears, 64 per cent.; large lymphocytes, 30 per cent.; transitionals, 6 per cent. The urine was acid, with a trace of albumin; the microscopic examination was negative.

The next day there was not much change in the condition. The child fretted a great deal, and lay with his legs in a froglike position (infiltration of tissue due to hypodermic cod liver oil?). A roentgenogram of the extremities showed mild rickets. Examination of the right eye showed the ulcer edge heavily infiltrated with leukocytes. The child seemed much soothed when the eye was compressed.

The general condition was improved, June 10. The child was very fussy, but behaved in a more natural manner. The first weight gain was noticed, the present weight being 15 pounds, 3 ounces (6.8 kg.). The right eye was sunken; the left eye showed a normal luster with the corneal reflex normal.

June 13, the cod liver oil by hypodermic was omitted, and one-half teaspoonful, three times daily, ordered.

June 15, the child fussed a great deal when disturbed, but this might have been due to the great amount of induration that was present at the points of injection of the cod liver oil. He was quiet unless disturbed. The general condition was improved. Examination showed no rigidity of the neck or of the extremities. The liver edge felt much softer on palpation. The mouth was clean and moist; the cracks in the lips had healed.

Eight days later, when quartz lamp exposure was begun, the temperature dropped from 102.2 to 98.2, and for the rest of the hospital stay rarely exceeded 100.

The condition remained stationary for about ten days. The child took food very poorly and vomited feedings if they were urged. The bowels remained normal, with a slight tendency to constipation. The child fussed a great deal, particularly if disturbed.

June 26, with no changes in diet, the infant gained in weight. After this, he became much happier, took feedings eagerly, and convalescence progressed without further event, as shown in the accompanying chart.

COMMENT

This case presents a number of unusual features. The appearance of keratomalacia three weeks after the apparent deprivation of fat soluble A constitutes a startlingly rapid development of the disease. While in animal experimentation such rapid development frequently obtains, there one is dealing with a different life cycle. One might assume some comparative diet deficiency during the time preceding the actual onset of the symptoms, but in this case the infant was said to have been in perfect health prior to the onset of the illness.

The study of the clinical history reveals in this instance a rather unusual manifestation of nervous symptoms. The first impression, on examination of

this patient, was the presence of disease of the nervous system. In the differential diagnosis, the following diseases were considered: erythredema, typhoid fever, tuberculous meningitis, scurvy and encephalitis. There was a very definite suggestion of cerebral irritation. In a study of the literature, this appearance of nervous symptoms has been not infrequently noted. As early as 1866, von Graefe,[16] who gave the first accurate description of the eye symptoms, mentioned, as general symptoms, pallor, diminished tonus, poor digestion and anorexia, but no striking cerebral symptoms. This was mentioned because of one case, particularly, in which the patient had convulsions and lethargy preceding death. Klebs [17] found an encephalitis on postmortem examination. Later observers discounted these early reports of encephalitis. Schulz [18] reported a case showing mild convulsions, the head in opisthotonos and marked rigidity of the neck. On postmortem examination, the brain was normal. Stephenson [19] noted opisthotonos in five out of thirty-one cases seen by him, but in one alone was there meningitis, as proved by postmortem examination. Bloch [8] describes the infants in his Type B classification as very fretful and sensitive to outside impressions. Stolte [20] not only observed hyperirritability, but noticed that some who recovered later had spasmophilia. Of Ross' [6] reported cases, opisthotonos and neck rigidity were noticed in Cases 3 and 4.

The outstanding symptoms of this patient were stupor, irritability on being disturbed, weight loss and other evidences of nutritional disturbance (pallor, doughy skin), dry inflamed mouth and cracked lips, fever, and the characteristic corneal ulceration. The liver seemed unusually large and firm when the child was first seen. The symptoms suggestive of meningeal disturbance persisted for a number of days. Besides the symptoms in the case report, it should be mentioned that transient strabismus was observed the day preceding hospital admission. Spinal fluid examination showed no pleocytosis nor increased globulin. There was slightly diminished sugar reduction. The child lay in the froglike position occasionally observed in scurvy. As the child had had intragluteal hypodermic medication, it was hard to rule this out as a factor in the position assumed.

The patient showed complete recovery, except for the loss of vision in the right eye. The response to the treatment with fat soluble A substance in this patient with keratomalacia was not nearly so striking as that often witnessed in scurvy following the use of orange juice. Despite the hypodermic administration of cod liver oil as suggested by Bloch, convalescence was slow. The absorption of cod liver oil thus given was very slow. It was three weeks after specific treatment was begun before the weight curve and the other symptoms showed appreciable change. It is very obvious that the appearance of the characteristic eye changes are ominous of more advanced nutritional disturbance than is apparent. Wolbach's [21] investigation leads one to believe that the changes undergone by the experimental subjects of this disease are more generalized than have heretofore been assumed. Wolbach found a considerable degree of atrophy in all of the epithelial tissues of the body, with ultimate keratinization of these tissues.

16. Von Graefe, A.: Hornhautverschwärung bei infantiler Encephalitis, Arch. f. Ophth. **12**: 250, 1866.
17. Klebs: Arch. f. Ophth. **12**: 250, 1866.
18. Schulz, R.: Beitrag zur Lehre von der Xerosis Conjunctivae und der infantiler Hornhautverschwärung, Arch. f. Ophth. **30**: 123, 1884.
19. Stephenson, S.: On Sloughing Corneae in Infants, Ophthalmoscope **8**: 782, 1910.
20. Stolte, K.: Ueber Keratomalazie, Klin. Monatsbl. f. Augenh. **68**: 739, 1922.
21. Wolbach, S. B., and Howe, P. R.: The Epithelial Tissues in Experimental Xerophthalmia, Proc. Soc. Exper. Biol. & Med. **22**: 402, 1925.

SUMMARY

1. The occurrence of keratomalacia in an infant following a diet restricted for therapeutic purposes constitutes an almost experimental production of the disease.
2. Besides the usual weight loss and nutritional upset, the infant presented pronounced meningeal symptoms.
3. The patient recovered following the administration of cod liver oil, given by mouth and hypodermically, and a diet of whole lactic acid milk.

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THE TREATMENT OF ERYTHREMIA WITH PHENYLHYDRAZIN *

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In a previous paper,[1] a case of polycythemia was reported which had been under treatment with phenylhydrazin hydrochlorid. Since the result in that case was so satisfactory, and since none of the other methods of treatment for this disease have been more than temporarily beneficial, it seems desirable to record the observations in four more cases treated with this drug in order that its actual value may be finally estimated. As I pointed out in the first paper, phenylhydrazin was used by Morowitz and Pratt to produce secondary anemia in animals, after which Eppinger suggested its use in the treatment of erythremia. Later, Taschenberg experimented with the drug in one patient. The results obtained in their cases were not successful, probably because the patients were in a late stage of the disease.

If phenylhydrazin proves of benefit in the treatment of the disease, it is important, because by the time patients with erythremia are compelled by their symptoms to ask a physician's advice, the problem is not so much one of diagnosis as of therapeusis. Previously, even with the combination of frequent bleedings and roentgen-ray or radium application, the certainty of being able to reduce the red cells and hemoglobin to somewhere within normal limits and to keep them there has been a difficult and sometimes impossible task.

The subsequent course of the case referred to (Case 3) can now be given, together with the results of treatment by phenylhydrazin of four other cases of erythremia.

The first two patients were treated by Dr. John L. Dorsey of Baltimore and, as far as I know, are the earliest ones to receive the drug in this country. He has kindly allowed me to include them in this report.

CASE 1.—A woman, aged 60, who had a true polycythemia of some years' standing, seen, April 13, 1923, complained of increasingly severe headaches and pains in the fingers. She presented a typical picture of the disease with a large spleen. The red blood count was 10,112,000; the white cells, 5,000, and the hemoglobin, 120 per cent. (Dare). During April, 1923, she was given four radium treatments with no apparent benefit and no change in the blood count. May 22, 1923, she began a course of phenylhydrazin in 0.1 gm. doses daily for twenty days. This was given in capsules by mouth.

Table 1 shows the dosage and the variations in the blood counts during treatment in this patient.

* From the Department of Medicine, Johns Hopkins Hospital.
1. Owen, Trevor: A Case of Polycythaemia Vera with Special Reference to the Familial Features and Treatment with Phenylhydrazine, Bull. Johns Hopkins Hosp. **35**: 258-262 (Aug.) 1924.

TABLE 1.—*Dosage and Variations in Blood Counts During Treatment in Case 1*

Date	Doses of Phenylhydrazin	Hemoglobin	Red Blood Cells	White Blood Cells	Polymorphonuclears, per Cent.	Comment
1923						
4/13	Irradiation 4 times in April, 1923	120	10,112,000	5,000	81	
4/27	.....	120	8,448,000	4,800	..	
5/11	.....	120	9,520,000	6,200	71	
5/22	0.1 gm. each day for 20 days ending June 10					
5/25	.....	120	9,200,000	4,000	80	
6/ 1	.....	120	7,760,000	2,600	..	
6/11	.....	120	7,600,000	.....	..	
6/19	.....	115	7,456,000	.....	..	
6/20	0.1 gm.					
6/21	0.1 gm.					
6/22	0.1 gm.					
6/23	0.1 gm.					
6/24	0.1 gm.					
6/25	0.1 gm.	105	5,808,000	5,600	72	
7/ 3	.....	80	5,120,000	5,200	68	Platelets increased
7/10	.....	85	4,384,000	7,000	82	
7/18	.....	75	3,904,000	5,200	..	
8/ 3	.....	95	4,640,000	3,200	..	
8/27	.....	105	4,972,000	5,600	79	
10/ 6	.....	110	5,712,000	4,800	77	
10/ 8	0.1 gm.					
10/ 9	0.1 gm.					
10/10	0.1 gm.					
10/11	0.1 gm.					
10/12	0.1 gm.					
10/25	.....	105	5,104,000	3,400	..	
11/28	.....	95	4,256,000	4,800	..	
12/21	.....	96	4,704,000	6,400	..	
1924						
2/14	0.1 gm. daily for 5 doses	120	6,528,000	10,000	77	
3/ 5	0.1 gm. daily for 5 doses	112	6,336,000	5,200	86	
3/18	0.1 gm. daily for 5 doses	110	5,840,000	4,200	84	
3/24	0.1 gm. daily for 5 doses	105	5,534,000	7,000	..	
4/ 2	.....	95	4,176,000	9,200	88	Occasional myelocyte
5/ 3	.....	98	4,616,000	5,400	..	
5/ 9	0.1 gm. daily for 5 doses					
6/17	0.1 gm. daily for 5 doses	118	6,064,000	8,400	..	
6/30	.....	100	4,320,000	5,800	81	
7/15	0.1 gm. daily for 5 doses					
7/28	.....	93	4,112,000	8,200	..	
8/15	0.1 gm. daily for 5 doses					
8/22	0.1 gm. daily for 5 doses					
9/ 4	.....	90	4,400,000	6,000	..	
9/18	0.1 gm. daily for 5 doses					
10/ 3	0.1 gm.					
10/ 4	0.1 gm.					
10/ 5	0.1 gm.					
10/ 6	0.1 gm.	105	4,688,000	6,000	83	
10/ 7	0.1 gm.					
11/15	0.1 gm. daily for 5 doses					
12/ 1	0.1 gm. daily for 5 doses	120	5,248,000	3,800	..	
12/22	0.1 gm. daily for 5 doses	110	5,904,000	7,800	..	
1925						
1/ 5	0.1 gm. daily for 5 doses	110	5,184,000	4,600	..	
2/ 1	0.1 gm. daily for 10 doses					
2/22	.....	90	5,680,000	.....	..	
3/ 1	0.1 gm. daily for 5 doses					
4/11	0.1 gm. daily for 5 doses	105	5,696,000	4,800	..	
4/30	0.1 gm. daily for 5 doses	105	5,088,000	3,800	..	
5/17	0.1 gm. daily for 5 doses					
6/ 9	0.1 gm. daily for 5 doses	108	4,960,000	5,000	..	

The facts to be particularly noted in the table are that the polymorphonuclear percentage was usually high; a mild secondary anemia occurred once, and there was always an exacerbation of the symptoms whenever the blood count rose.

It will be seen that 2.6 gm. was originally required to bring the blood down to normal, the count gradually falling for three weeks after the drug had been stopped. Three months elapsed before a resumption of treatment was necessary, after which the drug was given as indicated. In these twenty-five months, a total of 14.1 gm. of phenylhydrazin has been used, which is approximately 0.13 gm. a week. There have been no unpleasant symptoms from the drug, and now the patient is in good health.

CASE 2.—A widow, aged 60, who had consulted Dr. Dorsey, Dec. 5, 1923, because of pain in the chest, had had bad health for several years. Her husband died in 1903 of cirrhosis of the liver, and she had a nervous breakdown following this trouble. During this time, there was some numbness in the arms. Gradually, she had become more and more easily fatigued. She had an old sinus infection which was kept under observation. There was an appendectomy in 1917. In 1919, some neck ligaments were torn in a motor accident, and she was in bed for several weeks. She suffered from frequent headaches, which were relieved by codein. Several abscessed teeth were removed during the last few years. There was no history of hemorrhages. The family history was negative.

For a few weeks she had been having a sharp pain over the precordium. This did not radiate and was not related to exercise. It frequently woke her up at night with a feeling of oppression. Very frequently she would have a tense aching pain in the fingers.

The patient was a well nourished woman. Her face was slightly red, and the lips, hands and feet were definitely cyanotic. The fundi were normal; the veins not overfull. Around the margin of both disks, there were several areas resembling old hemorrhages or choroidal degeneration. The pharyngeal and buccal mucous membrane was fiery red. A good many dilated veins were present over the sternum. The lungs were clear. The heart was slightly enlarged, the left border being 14 cm. to the left of the median line, and the right border 4 cm. There was definite retromanubrial dulness. The heart sounds were normal except for a gallop rhythm. No murmurs were heard. The radial and brachial arteries were moderately thickened, and the systolic blood pressure was 170; diastolic, 125. The abdomen appeared normal, but the spleen was enlarged and reached to within 6 cm. of the umbilicus. The liver just reached the costal margin. There was edema of the feet. The reflexes everywhere were normal.

The urine varied in specific gravity from 1.010 to 1.020; a trace of albumin and an occasional hyaline cast was present. The phenolsulphonephthalein excretion was 59 per cent. in two hours. The blood examination showed: 8,342,000 red cells; 10,840 white cells, and 122 per cent. hemoglobin (Sahli). There was slight anisocytosis. In the differential count, there were 73.2 per cent. polymorphonuclears and 26.4 per cent. lymphocytes. The platelets were increased. The blood nonprotein nitrogen was 34.2 mg. per hundred cubic centimeters; sugar, 10 mg. per hundred cubic centimeters; uric acid, 3.49 mg. per hundred cubic centimeters; creatinin, 3.0 mg. per hundred cubic centimeters. The Wassermann reaction was negative. The stools were normal. Roentgen-ray examination of the chest showed a moderate enlargement of the heart, a dilated aorta, and a generalized nontuberculous infiltration of both lungs. The diagnosis was erythremia, arteriosclerosis and hypertension.

Table 2 shows the course of this case under treatment. It was carried out while the patient was up and about.

The noteworthy facts shown in Table 2 are: There was always a high polymorphonuclear percentage; a relatively slight leukocytosis occurred; the blood pressure fell to a normal level, and the liver became slightly larger.

In summary, this patient required a total of 12.4 gm. of phenylhydrazin in the seventy-seven weeks, an average of about 0.16 gm. a week, to keep the blood normal. A slight secondary anemia was produced on two occasions, but otherwise the course was uneventful.

CASE 3.—A man, aged 46, who had had erythremia probably for ten years, developed neurologic symptoms only five months before coming under observation in December, 1923. Imme-

diately before treatment was begun, the blood examination showed: hemoglobin, 145 per cent.; red blood cells, 7,612,000; leukocytes, 8,000. In the course of three weeks the patient was given 2.4 gm. of phenylhydrazin, with the result that his blood count became normal and he felt well. After discharge from the hospital, there was an exacerbation of his symptoms and another rise in the blood count. This was again easily reduced to normal after another course of the drug in May and June, 1924, during which time he received a total of 1 gm. Since then, a matter of twelve months, he has been followed very carefully and has remained perfectly well. Complete blood counts were made every two weeks for the first few months. As the hemoglobin at the end of this time was 105 per cent. and the red cells 5,500,000, 0.1 gm. doses were given twice in the last two weeks of July, once a week in August, and three times in September. Since then, he has taken 0.1 gm. once a week. On this small but regular dosage, the blood count remained normal until the end of 1924, when a gradual rise was noted. In December, 1924, the hemoglobin was 108; red cells 5,640,000, and the leukocytes 10,800. During January, February and March the counts were reduced slightly. During April, no phenylhydrazin was taken, but in May he took 0.1 gm. every five days and in spite of this, at the end of the month, the red cells were found to be 8,900,000, and the hemoglobin, 115. In June, he took 0.1 gm. every other day with little effect, and by June 19 the hemoglobin was unchanged and the red cells were 7,100,000. A fresh supply of phenylhydrazin was obtained, and he was given 0.1 gm. every day for ten days. This has resulted in complete recovery again, and at the present time of writing (July 29, 1925), the hemoglobin is 100, the red cells 4,360,000 and the white cells 10,000. Though this increase in blood count was accompanied by a return of some of the symptoms, the patient has not had to stop work at any time and has felt perfectly comfortable since beginning treatment.

TABLE 2.—*Dosage and Variations in Blood Counts During Treatment in Case 2*

Date	Doses of Phenylhydrazin	Hemoglobin	Red Blood Cells	White Blood Cells	Polymorphonuclears, per Cent.	Comment
1923						
12/ 5	.....	...	7,968,000	7,200	..	Blood pressure 170/125
12/ 6	.....	120	9,568,000	6,000	80	
12/13	0.1 gm. each day for 20 days ending Jan. 1, 1924					
12/19	.....	120	7,648,000	7,600	..	
12/24	.....	120	6,784,000	6,600	83	
12/29	.....	115	6,190,000	.....	..	
1924						
1/ 2	.....	108	5,648,000	13,800	89	
1/ 7	.....	90	7,200,000	13,700	85	
1/29	.....	90	5,500,000	10,800	76	Some headache; not so easily tired; down South
2/26	.....	...	5,600,000	9,900	75	
3/10	0.1 gm.					
3/11	0.1 gm.					
3/12	0.1 gm.					
3/13	0.1 gm.					
3/14	0.1 gm.					
3/25	.....	...	.....	.....	..	Precordial pain; two attacks
4/ 1	.....	85	6,200,000	9,300	80	
4/10	0.1 gm. daily for 5 doses					
4/18	0.1 gm. daily for 5 doses	120	5,312,000	20,000	85	Still tires easily; aching fingers; "ringing" in head; dizziness; returned from the South
4/22	.....	...	.....	.....	..	Blood pressure 120/70; two nosebleeds; one attack of precordial pain; no cyanosis
4/29	.....	95	4,544,000	9,000	..	
5/14	0.1 gm. daily for 5 doses	105	4,900,000	4,800	..	No pain; no epistaxes; less tired
5/28	.....	100	4,600,000	7,600	80	Severe headaches; one nosebleed
6/11	0.1 gm. daily for 5 doses	95	5,760,000	9,800	..	
6/24	.....	93	4,040,000	5,600	..	
7/ 8	.....	98	4,480,000	7,400	83	
7/22	.....	90	4,800,000	4,200	..	Temporary diarrhea; spleen, 3 F. B. below costal margin
8/ 1	0.1 gm. daily for 5 doses					
9/ 2	.....	96	5,250,000	12,000	..	
9/ 9	0.1 gm. daily for 5 doses	...	.....	.....	..	Stronger; a few headaches
10/ 9	0.1 gm. daily for 5 doses	108	5,680,000	8,600	..	Blood pressure 138/95; much better
10/20	0.1 gm. daily for 5 doses	103	5,360,000	8,400	90	
10/27	.....	100	4,624,000	7,400	81	Numbness and tingling of toes after walking
11/10	0.1 gm. daily for 8 doses					
11/26	.....	78	3,300,000	7,500	93	Numbness of feet; morning headaches
12/ 4	.....	70	3,760,000	7,200	..	
12/16	.....	90	4,336,000	6,800	85	Feels better
1925						
1/ 5	.....	100	4,736,000	8,400	..	Went South
1/15	0.1 gm. daily for 5 doses					

TABLE 2—*Continued*

Date	Doses of Phenylhydrazin	Hemoglobin	Red Blood Cells	White Blood Cells	Polymorphonuclears, per Cent.	Comment
2/ 9	.....	104	7,640,000	8,400	83	Felt badly for a week; headaches severe; becoming red
2/11	0.4 gm.					
2/12	0.4 gm.	...	6,700,000			
2/13	0.2 gm.					
2/14	0.2 gm.					
2/15	0.1 gm.					
2/16	0.1 gm.					
2/17	0.1 gm.	100	6,240,000	16,400	84	
2/18	0.1 gm.					
2/20	.....	88	4,768,000	.....	..	Bronchitis; palpitation; dizziness
2/25	.....	96	4,808,000	13,375	84	
3/10	.....	90	5,574,000	13,000	86	
3/13	0.1 gm. daily for 5 doses					
3/25	.....	94	5,700,000	13,000	86	
3/26	0.1 gm. daily for 5 doses					
4/10	0.1 gm. daily for 5 doses					
4/16	.....	95	.....	7,800	..	Returned from the South; spleen, 4 F. B. below costal margin; liver, 2 F. B.; no cyanosis; blood pressure, 130/80
4/25	0.1 gm. daily for 5 doses					
5/ 5	.....	95	4,480,000	9,000	..	
5/10	0.1 gm. daily for 5 doses					
5/19	.....	81	4,170,000	7,800	..	
5/25	0.1 gm. daily for 5 doses					
6/ 6	.....	80	3,200,000	8,800	..	

When these patients are studied by daily counts during the administration of the drug, interesting and important changes take place. The next two patients, who were in the Johns Hopkins Hospital for treatment, illustrate this point.

CASE 4.—S. M., a Russian laborer, aged 62, was admitted Oct. 14, 1924, presenting all the features of a severe grade of polycythemia. His complaints were redness of the hands, headache, weakness and a sensation of internal heat. He was a brilliant purplish red, with dilated veins over the head, neck and arms and to a lesser extent in the legs. He had a large colloid goiter, considerable emphysema, a slightly enlarged heart, moderate thickening of the arteries, and a blood pressure of 160 systolic and 100 diastolic. The liver and spleen were just palpable. There was slight dyspnea and some sweating of the hands and feet. The blood showed 10,000,000 red cells, 8,000 leukocytes and 123 per cent. hemoglobin.

Chart 1 shows the effect of the treatment. This was started, October 31, with daily doses of 0.2 gm. of phenylhydrazin. After twelve days this was discontinued, and urobilinuria, an increased leukocytosis, and a gradual fall in red count and hemoglobin took place. However, this proved to be only of temporary duration, and after a rise in counts another course of the same daily dosage was begun. In three or four days, the hemoglobin and red cells showed a rapid fall. The urobilin became greater, the spleen increased in size and on one day there was pain and a friction rub over it. The drug was not stopped altogether, but the dose was cut down to 0.1 gm. a day for three days, and then discontinued. In this

second course, 1.9 gm. had been given. For two weeks, the count kept on falling and reached 5,300,000 red cells and 88 per cent. hemoglobin. During this process, the patient's color became normal, the dyspnea disappeared, and he felt better than he had for many years. He was discharged two months after entry, the blood count then showing 6,000,000 erythrocytes and a hemoglobin of 90 per cent. The blood pressure had fallen to 140 systolic and 74 diastolic.

Supervision of this case could not be carried out as carefully as one would have liked because the patient lived out of the city. At the first, inadequate amounts were given, and in seven weeks with 0.2 gm. a week, the hemoglobin rose to 120 per cent., the red cells to 7,744,000, and the leukocytes to 20,000. During the next two weeks, 1.6 gm. was given with a most satisfactory result. The hemoglobin fell to 88 per cent., the red cells to 6,760,000 and the white cells to 7,000. From that date and for a period of nine weeks, a total of 2 gm. of phenylhydrazin was taken. At the end of that time, May 1, 1925, the hemoglobin was 106 per cent., the red cells 5,724,000 and the white cells 20,000.

In summary, 4.3 gm. of phenylhydrazin was required originally to reduce the blood count to normal. Five weeks after this course was completed, treatment was started again and for seventeen weeks an average weekly dose of approximately 0.3 gm. was necessary to keep the blood count about normal.

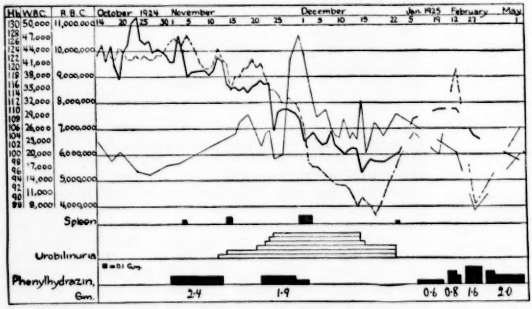


Chart 1 (Case 4).—Effect of doses of phenylhydrazin on blood count: heavy line, red blood corpuscles; light line, white blood corpuscles; broken line, hemoglobin.

CASE 5.—A man, aged 56, admitted to the Johns Hopkins Hospital, March 19, 1925, had had symptoms for four or five years and complained of attacks of pain in various parts of the body. He was the picture of a polycythemia of a moderate grade. The hemoglobin was 114 per cent.; the red cells 9,224,000 and white cells 18,200; polymorphonuclear cells, 80 per cent. The liver was just palpable, and the firm spleen was three fingerbreadths below the costal margin. The systolic blood pressure was 167; diastolic, 110. He weighed 157 pounds (71.2 kg.). Chart 2 shows the course of Case 5 under treatment. As in Case 4, beginning March 22, 0.2 gm. of phenylhydrazin was given daily for fifteen days, a total of 3 gm. A week after this was started, the hemoglobin and red cells were decreasing rapidly, and the leukocytes were rising. The leukocytosis is important and will be discussed below. After the drug was stopped, a further increase in the leukocytes took place, reaching 59,800 nine days later. As will be seen, a moderate secondary anemia was produced just after this point, the hemoglobin going to 45 per cent., and the red cells to 2,800,000. During this process, jaundice developed, the spleen became smaller, and there was a slight urobilinuria. There was also fever of from 99 to 100, which twice reached 101. The patient felt very weak and lost his appetite completely. There was a persistent cough, and it was thought that he had a bronchitis. Soon after the leukocytosis began to subside, the red cells and the hemoglobin commenced to rise. This did not occur until two weeks after the last dose of phenylhydrazin. On discharge, seven weeks after the first dose, the hemoglobin was 68 per cent., the red cells 4,144,000, and the leukocytes 16,800. The systolic blood pressure was 136; diastolic, 80. The patient was feeling much stronger, was free of pain and had a good appetite. A week later, May 18, the hemoglobin was 80 per cent., the red cells 4,800,000, and the leukocytes 22,000. He was told to have blood counts made every two or three weeks, and as soon as the hemoglobin reached 100, he was to begin taking 0.3 gm. of phenylhydrazin a week, but he was to stop if the leukocytes rose or the hemoglobin fell below 90.

COMMENT

Clinical Course.—An examination of the charts will make it clear that the use of this drug presents two problems. One of these has to do with the rapidity with which the drug can be given, and the other with the duration of its administration. Excessive doses will cause secondary anemia, which, even though temporary, may be sufficiently severe to be alarming. As the drug is not excreted very rapidly, the effect is cumulative. In Cases 1, 2, 3 and 5, the red cell destruction continued for over two weeks after the drug was stopped, while on the other hand, in Case 4 (Chart 1), the first 2.4 gm. was not sufficient to be effective.

In the repeated studies that were made in Cases 4 and 5 shown in Charts 1 and 2, the first effect of the drug which could be observed was an increase in the leukocytes. This occurred before any changes were noticed in the number of red blood cells or in the percentage of hemoglobin. Referring to Chart 1, it can be seen that, on admission, the leukocytes were 23,000, while at rest in bed during the next twelve days, the leukocytes fell to 15,500, the red cells at the same time increasing to 11,300,000. Phenylhydrazin was then begun, and during the first course of the drug the leukocytes rose gradually from 17,200 to 30,000. Soon after this increase in the white blood cells had begun, the hemoglobin and red cells were noticed to fall, and urobilin appeared in the urine. After the drug was stopped, the counts tended to revert to their previous levels. When a second course of phenylhydrazin was begun after an interval of only nine days, there was an abrupt rise in the white count from 19,000 to 47,500. When the dose was reduced, the leukocytes fell again to about 30,000 and the red cells increased.

In Case 5 (Chart 2), twelve days after the first dose, there was a leukocytosis of 24,000, and nine days after the last dose this had increased to 59,000 and a marked decrease in the red cells and hemoglobin followed. It was observed that, in the two cases when daily counts were made, this rise in the leukocytes always preceded the fall in the red cells and hemoglobin, and that the greatest leukocytosis came before the greatest drop in the hemoglobin and red cells. This is also seen most strikingly in Chart 2 of Case 5. With this increase in the leukocytes and the rapid fall of the hemoglobin and red cells, there was a moderate icterus with a slight urobilinuria in Case 5, and a marked urobilinuria with jaundice in Case 4. It was also noticed that, although the leukocytes fell to a lower level after the drug was discontinued, they did not reach normal figures until the red cells and hemoglobin had begun to rise again. In Cases 1 and 2, a leukopenia was found with smaller

doses, but when larger amounts were given a leukocytosis occurred. These observations seem to point clearly to the fact that on the administration of phenylhydrazin, a leukocytosis preceded the fall in hemoglobin and red blood cells, and that the reduction of hemoglobin and red blood cells which follows is roughly proportional to the leukocytosis. It is necessary, therefore, to follow the changes in the leukocyte counts when the drug is being given, for an increase in the white cells is an indication for stopping the drug.

The question of the possible causes of the leukocytosis is an interesting one. One might conceive of the leukocytosis as being due to a direct effect of the drug on the bone marrow, or hematopoietic tissue, but such an explanation seems unlikely, in spite of the fact that the bone marrow is hyperplastic in animals poisoned with the drug. A more likely explanation of the leukocytosis might be that it is a compensatory mechanism due to injury which phenylhydrazin causes to the cells of the liver, and certainly to the erythrocytes. With phenylhydrazin administration, a destruction of red blood cells is evidenced by a decrease in their numbers and by urobilinuria, and when large doses are employed, these are so greatly increased with jaundice accompanying them that one suspects liver cell destruction as well. A similar destruction of tissue and especially of the liver may account for the leukocytosis seen in certain cases of acute poisoning with such substances as mercuric chlorid, lead, arsenic and *Amanita phalloides*, and in some cases of acute yellow atrophy. These substances also injure tissue cells, and the leukocytosis may indicate a certain degree of actual necrosis.

In animal experiments with the hydrazin compounds, extensive damage to the liver is known to occur. Wells,[2] studying the organs of dogs, cats and guineapigs to which he had given toxic doses of hydrazin, and Underhill and Kleiner,[3] in similar experiments, found very constant changes. The liver alone was usually affected. It was friable and very yellow. In every case, there was fatty degeneration of the cytoplasm of the parenchymatous cells beginning at the center of the lobule and progressing outward. He saw no focal necroses. The statement is also made that the hydrazin had apparently no effect on the red cells because no abnormal pigmentation of the liver cells, spleen or lymph glands was present. Later, Underhill [4] gave dogs large doses of the various hydrazin compounds. No details are recorded, but the following effects were noted: In a 10 kg. dog given a dose of 500 mg. of phenylhydrazin, the liver was of a peculiar chocolate color and the spleen enormously enlarged and dark purple.

Methylphenylhydrazin had no effect on rabbits, and in a dog weighing 4 kg., which received 200 mg., the organs were uninjured. The same was true of diphenylhydrazin and semicarbazid. Bodansky [5] made a study of the same kind, estimating the amount of liver damage by sugar tolerance tests. His observations are in agreement with the previous ones, but examinations of the organs are given in more detail. Unlike Wells, he did find areas of necrosis in the liver. The importance of these experiments lies in the fact that some of the derivatives of hydrazin are less destructive to liver cells than others, though they are equally effective in destroying red blood cells. With the free base, a dog weighing 10.4 kg. died, after 0.294 gm., with fatty degeneration of the liver and areas of necrosis. Hydrazin sulphate was slightly more toxic. With phenylhydrazin, 120 mg. given to a dog weighing 5.9 kg. produced deficient liver function. There was marked anemia. At necropsy there was liver cell damage with marked hyperplasia of the spleen and bone marrow. A little fatty degeneration was found in the kidneys. Methylphenylhydrazin was less harmful both to blood and to liver. Diphenylhydrazin had no effect. Acetylphenylhydrazin, on the other hand, possessed the power of reducing the number of red blood cells, though not quite to the same degree as phenylhydrazin, but did no damage to the liver. The dog was given two injections (0.71 gm.) and became very anemic, but the liver efficiency was not lowered and the organ was normal at necropsy.

It seems, therefore, from these and similar observations that we may find other derivatives of the hydrazin

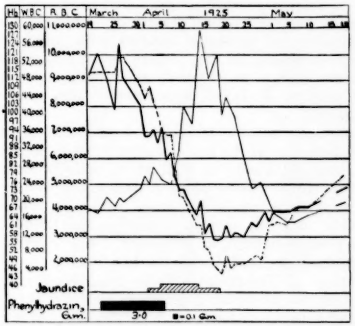


Chart 2 (Case 5).—Effect of doses of phenylhydrazin on blood count.

series which will be more suitable to use for therapeutic purposes than phenylhydrazin. Acetylphenylhydrazin, for instance, seems preferable because in animals it is less toxic to liver substance, while retaining the power of destroying red blood cells.

Whether repeated small doses of phenylhydrazin, such as were given to patients, will or will not be deleterious to the liver cannot be said yet. It is conceivable that a cirrhosis may ensue, but whether this will be any greater than that which ordinarily occurs needs further study. Levi,[6] from the study of his case which came to necropsy, decided that there is danger in the use of the drug because liver cirrhosis may be produced. He is not quite sure whether this is due to the action of phenylhydrazin directly, or occurs secondarily from the increased blood destruction. His patient was a man of 50, of nonalcoholic history, who received 7.5 gm. of phenylhydrazin during a year and a half. This was given in 0.2 gm. gelatin capsules once a day in five courses, three of 1.5 gm. each, one of 2 gm. and one of 1 gm. Each course was begun when the hemoglobin reached 110. There was jaundice and urobilinuria during these courses of the

2. Wells, H. G.: The Pathological Anatomy of Hydrazine Poisoning, J. Exper. Med. **10**: 457-464, 1908.

3. Underhill, F. P., and Kleiner, I. S.: The Influence of Hydrazine upon Intermediary Metabolism in the Dog, J. Biol. Chem. **4**: 165-178, 1908.

4. Underhill, F. P.: Studies in Carbohydrate Metabolism, J. Biol. Chem. **17**: 295-298, 1914.

5. Bodansky, M.: The Action of Hydrazine and Some of Its Derivatives in Producing Liver Injury as Measured by the Effect on Levulose Tolerance, J. Biol. Chem. **58**: 799-811 (Jan.) 1924.

6. Levi, Ernst: Ztschr. f. klin. Med. **100**: 777-784, 1924.

drug. The patient died of an intercurrent infection (erysipelas). The necropsy findings showed a much enlarged spleen, rich in leukocytes and with extensive deposits of blood pigment. The liver was of medium size, the surface slightly irregular, and on section was of a peculiar yellowish color. Microscopically, the interlobular connective tissue was greatly increased and infiltrated, with round cells. There were many centers of regeneration. The bone marrow was very hyperplastic, the myelocytes obliterating the normoblasts. He discussed at length the probabilities of the various factors that may cause the cirrhosis. The fact that there are cases of polycythemia with and without cirrhosis argues against the cirrhosis as a primary lesion, and he thinks that most likely the blood destruction injures the liver and that the natural course of events is only exaggerated by the artificial blood destruction produced by phenylhydrazin. He concludes by making the very fair statement that, whether the patient is left to deal with his plethora as best he can, or whether phenylhydrazin is added to assist his own resources with the chance that cirrhosis follow, is a question for each particular patient and his physician to decide. However, the latter risk is unknown and may be relatively small as compared to the risks that we know are run without treatment. Moreover, since the majority of untreated patients with polycythemia develop cirrhosis, if they escape other complications, it seems logical to conclude that their own inadequate method of blood destruction is not necessarily successful.

From these considerations and from the observations on the behavior of the leukocytes in the patients treated with phenylhydrazin, as well as in patients suffering from the toxic effects of other poisons, it is felt that the occurrence of leukocytosis in these conditions probably indicates actual destruction of tissue cells. With phenylhydrazin, in particular, the liver cells may be destroyed as well as the red blood cells.

Method of Treatment.—From these studies, it has been possible to devise a tentative plan for the treatment of patients suffering with erythremia. It is obviously most important to control the treatment by careful and frequent blood counts with especial reference to the changes in the leukocytes. This is absolutely essential when large doses of phenylhydrazin hydrochlorid are being administered.

It is safe to begin treatment by giving 0.2 gm. of phenylhydrazin a day for three or four days, and then 0.1 gm. every day until the leukocytes increase in number or the hemoglobin falls below 100 per cent. When the leukocytes rise or the hemoglobin falls, which may occur in two, three or four weeks' time, 0.1 gm. may be given every second or third day, or the drug may be stopped altogether. When the rise in leukocytes is marked, it is safest to discontinue the drug entirely. So long as the leukocytes remain high, even though this persists two weeks or more, the drug should be withheld, in spite of the fact that the red cells and hemoglobin may not fall. It is unlikely that this will happen. If the hemoglobin and red cells begin to rise or the leukocytes begin to fall, the dose should be started again or increased in frequency. Gradually the interval between doses can be lengthened, and usually 0.1 gm. once a week will suffice to keep the blood count within normal limits.

Symptoms Under Treatment.—So far, no ill effects have been noticed in these patients. Two of the patients complained of slightly disagreeable sensations after taking the capsules. In one, there was a sense of flushing and for some days slight fever. The other patient complained of a mild oppressive sensation in the neck. In two of the cases, there was slight hematuria during the administration of the drug; but as this is not an uncommon symptom of the disease and since it ceased in both patients while they were still taking the drug, it is not clear that the phenylhydrazin was the actual cause of the hematuria. Two of the patients had acute bronchitis during the treatment.

In two of the patients, the spleen increased in size at the height of the blood destruction (Cases 3 and 4) while in another (Case 5), there was a diminution in the size of the spleen. In the latter case, the liver seemed to enlarge as the spleen diminished in size. A most satisfactory result has been the reduction of the hypertension which was present in three of the patients. In Case 2, the blood pressure fell from 170/125 to 130/80; in Case 4, from 160/100 to 140/74; and in Case 5, from 167/110 to 136/80.

Changes in the Urine.—Evidently, the phenylhydrazin in some form is excreted in the urine. Soon after the drug is started, the urine, when allowed to stand, turns very dark, almost black. We do not know in what form it is eliminated. The phenolsulphonephthalein excretion in two of the patients was estimated before and during treatment, but no increase was found during the administration of the drug. Levi [6] found hematoporphyrinuria on one occasion in his case.

CONCLUSIONS

1. Phenylhydrazin hydrochlorid acted beneficially in five cases of erythremia. This was evidenced by the uniform decrease of the hemoglobin and red blood cells, a great improvement in all the symptoms and a reduction in the blood pressure of three patients with hypertension.

2. One of the early effects of the drug is a leukocytosis, noted in two cases.

3. The occurrence of a leukocytosis is a highly important index in controlling the treatment.

4. The possibility of danger, because of the toxic action of the drug on the liver, may perhaps be overcome by substituting another of the hydrazin compounds.

Orthostatic Albuminuria.—The connection between orthostatic albuminuria and a past attack of scarlatinal nephritis has been previously suggested. Bass, in 1912, gave an abstract of three papers dealing with the subject. Weiss-Eder allowed forty children with scarlet fever to kneel in a position of lordosis, with the result that twenty-three developed an albuminuria. The writer concludes that children convalescent from scarlet fever are not more disposed to nephritis than other children, and that such children, having nephritis, often show an orthostatic albuminuria, "which may be due to weak muscles." Nothman found that in twelve out of thirteen cases of scarlet fever in which at an earlier date the lordotic experiment was positive, albumin appeared later in the urine, but that in those in which lordosis did not cause albuminuria, albumin never appeared spontaneously later on. He concludes that, just as nephritis may terminate in orthostatic albuminuria, so conversely an orthostatic albuminuria may be the first sign of nephritis. Schick says that orthostatic albuminuria following scarlet fever has a good prognosis, and refers to Jehle, who examined seventeen children in various stages of scarlet fever.—Russell, J. W.: *Lancet*, Oct. 3, 1925.

Clinical Notes, Suggestions and New Instruments

A CASE OF TULAREMIA

W. L. BROWN, M.D., AND C. P. BROWN, M.D., EL PASO, TEXAS

A. L., a man, aged 58, a locomotive engineer, while oiling his engine preparatory to leaving El Paso at 8 p. m., Aug. 17, 1925, was bitten by an insect just above the prominence of the left cheek, three-fourths inch below and to the left of the eye. He noticed that it was an unusual bite, and thought it must have been made by a very large mosquito.

He made his run and by midnight began to have swelling in the region of the bite and pain in the glands on the corresponding side of his neck. He slept the following day. When he got up at 5 p. m., the glands were much swollen and were very tender and painful; there was considerable swelling at the site of the injury. He had no appetite and thinks he had fever at that time.

He brought his train back to El Paso that night, but the following morning he ached all over as if he had grip, was chilly, had fever, and the glands were painful. He took a cathartic, put cataplasma kaolini on the site of the injury and went to bed for his day's sleep. He awakened, sweating and chilling, during the day.

We saw him at our office on the fourth day, at which time he looked as if he had been sick a month. He was still feeling very bad and had a temperature of 102; the glands were sore, and he was having drenching sweats when he slept and all the symptoms of an acute infection. At the site of the injury there was a hard papular area one-fourth inch in diameter; it was raised, and the apex indicated that it was suppurating.

On removal of the skin covering this area, it showed evidence of a necrotic core. This core did not loosen and fall out until the eighth day, when it came away leaving a cavity so clear cut that it looked as if it had been punched out. The walls of this cavity were considerably above the surface of the skin, and the surrounding area was still very hard. There was no involvement of the eye. There seemed to be no question from the beginning that the insect bite was the cause of the illness. On the third day of our care we suspected tularemia, as the clinical picture was typical.

August 29, a blood examination showed white cells, 11,500, with 75 per cent. polymorphonuclears. Typhoid, paratyphoid A, and paratyphoid B agglutinations were negative. The urine was normal. September 1, two guinea-pigs were inoculated subcutaneously, one from a swab, and the other from scrapings of the ulcer. Both pigs died, September 5, within a few hours of each other, and the postmortem examinations showed typical lesions in the spleen and the liver, as described by Francis.[1]

The liver and the spleen from these dead animals were mixed with food and given to two other guinea-pigs which were confined in the cage in which the former had died. After a week of living in this cage and eating this food, they developed no symptoms.

September 12, agglutination for tularemia was positive in a dilution of 1:320. October 2, a suppurating cervical gland was opened, and two guinea-pigs were inoculated with this pus—from which, unfortunately, no cultures or smears were made—and twenty days later they showed no signs of ill health. On the nineteenth day following the inoculation, one of the pigs gave birth to a normal baby. This experiment would suggest that the pus did not contain *Bacterium tularense*. October 3, agglutination tests were positive in a dilution of 1:2,560, as reported by the U. S. Public Health Service.

The diagnosis in this case was made clinically on account of the history of an insect bite; enlargement and pain in the glands in the drainage area within twelve hours, with all the symptoms of a serious, acute, septic condition within forty-eight hours, especially characterized by chills and drenching sweats, and rapid loss of weight. Our patient lost 16 pounds (7.3 kg.) within eight days. Also, the local lesion was entirely characteristic, a hard papule one-fourth inch at the base, much elevated, and with extreme induration, the core falling out on the eighth or tenth day, leaving a clear cut, punched out hole.

The suspension of dead bacteria for the agglutination tests was secured from the U. S. Public Health Service, Washington, D. C., by Dr. John W. Tappan, United States public health officer, El Paso, and the laboratory work was done by Dr. Willis W. Waite, El Paso.

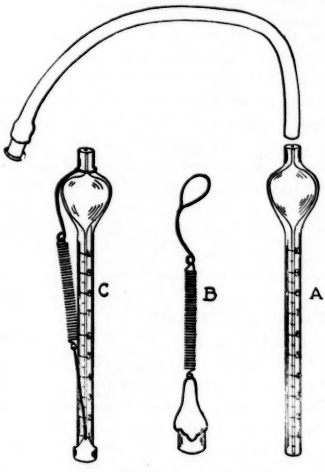
Roberts-Banner Building.

1. Francis, Edward: Tularemia, J. A. M. A. **84**: 1243 (April 25) 1925.

SEALING DEVICE FOR HEMATOCRIT*

C. M. VAN ALLEN, M.D., NEW HAVEN, CONN.

An hematocrit that has proved satisfactory because of its accuracy and simplicity and the small amount of blood required has already been described.[1] Recently, the efficiency of the instrument has been improved, by the substitution of a mechanical sealing device for the rubber band originally employed for the purpose of closing the lower end of the hematocrit during centrifugalization.



Sealing device for hematocrit.

The complete apparatus in its improved form is shown in the accompanying illustration. The hematocrit (*A*) consists in a capillary bulb pipet about 10 cm. in length. The shaft, from the lower extremity to the bulb, is graduated in a scale of 100 parts, the bore beneath this scale being of uniform caliber. A rubber mouth tube is fitted above the bulb. The sealing clip (*B*) is constructed of three parts: a wire loop to fit over the bulb; a metal cup, rubber cushioned, to enclose the end of the shaft, and a coil spring to retain the clip in place (*C*). The cushion is a rubber disk, such as may be cut from a broad rubber band, and is renewed readily. This clip fits also the standard hemocytometer pipet and is of use in conveying the filled pipet from a distance to the laboratory. Directions for the use of the hematocrit will be found in the papers referred to above.

* From the Laboratories of the Rockefeller Institute for Medical Research, New York.

* The hematocrit and sealing clip may be obtained from Arthur H. Thomas and Company, Philadelphia.

1. Van Allen, C. M.: An Hematocrit Method, J. A. M. A. **84**: 202 (Jan. 17) 1925; J. Lab. & Clin. Med. **10**: 1027 (Sept.) 1925.

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION

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SATURDAY, DECEMBER 26, 1925

LEAD POISONING

Within the brief period of thirteen years prior to the World War, more than 8,000 cases of plumbism with nearly 400 deaths were reported to the British Home Office as arising in the trades of the comparatively small population of Great Britain. Lead poisoning has existed from time immemorial. Students of the subject have ventured the statement that as little as 1 or 2 mg. of lead taken into the body each day is likely to lead to chronic poisoning. This represents a content of only one part of lead in 70,000,000 in the body of an adult of 70 kg. body weight. We cannot contemplate such possibilities without an appreciation of their seriousness. Guidance in combating the evil is eminently desirable.

It is therefore a fortunate circumstance that a group of investigators [1] working at the Harvard School of Public Health and the medical clinic of the Massachusetts General Hospital has been enabled to undertake a detailed study of the varied manifestations of a preventable hazard. The report of these investigators, recently made available, though reviewing much that has already been well established or clearly recognized, nevertheless serves to emphasize some of the factors—chemical, biologic, pathologic and clinical—that deserve emphasis at this time. The report makes evident anew that the route by which lead enters the organism most rapidly is the respiratory tract. Hence, the exposure to be most guarded against is the inhalation of finely divided particles. Next in danger comes the continued ingestion of lead compounds, and last of all, the possible slow cutaneous absorption in persons with tender, irritated or broken skin.

According to the Harvard studies, differences in distribution are found early in lead absorption, dependent on the route by which lead has entered the organism. Following absorption from the gastrointestinal tract, the liver contains a relatively high concentration of lead as a result of the retention of this toxic substance, which it removes from the portal blood. From the lung or subcutaneous tissue, on the contrary, lead enters the general circulation; hence, only a small portion of it is carried directly to the liver. Once in the general circulation, the lead is distributed throughout the organism. Since the skeleton is the only tissue to retain permanently any significant amount of lead, an ever increasing percentage of the total absorbed lead comes to be stored in the bones. After absorption has ceased, this selective localization of lead becomes practically complete. While stored in such a deposit the lead is apparently harmless, since the symptoms of plumbism are noted only when lead is generally distributed throughout the organism, as a result either of recent absorption from an external source or of mobilization of a skeletal store.

On the basis of the thesis that, after absorption into the general circulation, lead is either stored in the skeleton or eliminated from the organism by excretion, the problem of treatment may be more rationally approached. Obviously, during severe intoxication it would be wise to facilitate storage of lead rather than rapid mobilization of the element, because the latter might increase the "toxic episodes." In this connection the Harvard toxicologists have reported an important analogy between the metabolism of calcium and that of lead. Various decalcifying agents have been shown to increase the lead output. Conversely, conditions favoring calcium retention tend toward a complete storage of lead in the bones. Consequently, during periods of danger it is advised to facilitate storage of lead by the ingestion of ample calcium, rather than to set the lead free. After the acute toxic manifestations have passed, elimination of lead may be accelerated by a low intake of calcium, acids and their ammonium salts, sodium bicarbonate and potassium iodid. Caution should be exercised in using these forms of treatment on patients with nephritis.

Chemical studies have indicated that the products of the reaction between secondary sodium phosphate and a lead salt are tertiary lead phosphate and acid. This lead salt is very stable at the normal hydrogen ion concentration of the body, but is sensitive to slight changes in acidity and passes into the more soluble di-lead salt readily. The marked solubility of lead and lead oxid in such body fluids as blood plasma may account for the high incidence of lead poisoning among workers exposed to lead fume and lead oxid dust. Both gastric and pancreatic juice may transform insoluble lead salts into more soluble forms and may therefore cause an increased absorption of lead compounds. Lead is probably transported in the blood stream in the form of colloidal lead phosphate, and deposited in the bone tissue as tertiary lead phosphate. Tertiary lead phosphate is sensitive to changes in acidity, particularly to variations caused by such acids as lactic; this may in part account for the fact that

1. Aub, J. C.; Fairhall, L. T.; Minot, A. S., and Reznikoff, P.: Lead Poisoning, Medicine 4:1 (Feb.-May) 1925.

the concentration of lead in muscle is low and in bone tissue is high. The sensitiveness of tertiary lead phosphate to changes in hydrogen ion concentration may be an important factor in the frequent development of acute lead intoxication following acute infection or acidosis. It is generally agreed that by far the greater proportion of eliminated lead leaves the body with the feces. This is true of various other elements, such as calcium and iron. Some of them use the pathway of the bile as well as the gastro-intestinal epithelium. Small amounts of lead usually escape simultaneously through the kidneys. As one cannot readily determine whether fecal lead has actually been absorbed and reexcreted into the bowel, or merely has passed unchanged through the alimentary canal, the detection of lead in the urine is of greater clinical and diagnostic significance than the proof of its presence in the stools.

The sources of lead poisoning are manifold. Plumbism is a menace of many well recognized trades. There are, however, unsuspected byways through which lead may find its way into the body. One of these is contaminated alcoholic liquors. Instances of lead poisoning from the consumption of "moonshine" whisky evidently contaminated by the stills have been reported.[2] Among eighteen samples of liquor known to be used by saturnines, all except two showed the presence of lead. Inappropriate apparatus used in the manufacture and storage of wines and distilled liquors account for these disconcerting findings. The widespread use of containers glazed with lead compounds and the careless use of leaden or soldered containers in the preparation of home-made beverages are, therefore, matters of concern from the point of view of health.

A NEW FEATURE OF VITAMIN DEFICIENCY

Despite much investigation during the last decade as to the nature of deficiency diseases, little more has been done than to scratch the surface. Rapid additions to our knowledge, such as the spectacular discovery that pure cholesterol can be given vitamin activity through the influence of ultraviolet radiation, continue to be announced. Information that most concerns the practicing physician, namely, the extent to which minor degrees of vitamin deficiency modify human health, is still lacking, however. What relation, if any, has vitamin deficiency to resistance to infection, to fatigue, to well being? Most of the investigation of vitamins up to the present having been carried out in the laboratories of the physiologists and biochemists, the anatomic changes resulting from dietary deficiency have perhaps not yet been investigated as much as may be profitable. At least that is the inference that may be drawn from the interesting observations just reported by a pathologist, Wolbach of Harvard, on the effects of protracted deprivation of fat-soluble A vitamin.[1]

It has long been known that changes occur in the eye, the so-called xerophthalmia or keratomalacia, associated with drying, hornification and infection of the cornea, which have been considered to be characteristic of a deficiency of fat-soluble A. Wolbach and Howe find, however, that the eye changes are by no means the only anatomic effects of this deficiency, for they are only a part of a remarkable, widespread tendency for transformation of secreting and ductal columnar epithelium, or of transitional epithelium, into the heavily hornified type of epithelium normally serving for protective purposes. This metaplasia is found not only in the cornea and parocular glands, but even more extensively in the entire respiratory tract, from nose to bronchi; it is also found in the lining epithelium of the urinary passages, the salivary glands, the prostate, the uterus and oviducts, and the pancreatic ducts. Besides this tendency to keratinization there occurs a marked loss of fat, despite the presence of an abundance of fat in the animal's diet, and atrophy of glandular organs, especially the testes, spleen and salivary glands; but these atrophic changes are produced also by a deficiency of some of the other vitamins. Necropsy in a single case of deficiency of fat-soluble A in a child [2] has revealed hornification of the epithelium in the lungs, uterus, submaxillary glands, pancreatic ducts and renal pelvis, so that this effect seems to be a general one.

The process of keratinization begins in numerous foci, apparently independent of local infection or trauma, and spreads out from these, sometimes the hornified cells underlying for a time the original epithelium, which eventually is lost. Sometimes this process becomes so extensive that the hornified masses obstruct ducts, so that cysts are produced, and in the lungs bronchiectatic cavities form. A feature that is certain to arouse discussion is the resemblance at times of this proliferation of hornifying epithelium to the early stages of carcinoma formation, especially since occasionally metaplasia of columnar to stratified epithelium precedes carcinoma formation, particularly in the lungs and renal pelvis. Is it possible that a deficiency in fat-soluble A has anything to do with the formation of squamous cell carcinoma? At first glance this seems unlikely, for in general good nutrition seems to be more favorable for cancer formation than poor nutrition. But as yet we know too little about the relation of nutrition to cancer, or about the relation of vitamin content of human food to vitamin absorption and utilization, to speak with any assurance on this question. In any event, the demonstration that an absence of something in the food

2. Vaughan, W. T.: Lead Poisoning from Drinking "Moonshine" Whisky, J. A. M. A. **79**: 966 (Sept. 16) 1922.

1. Wolbach, S. B., and Howe, P. R.: J. Exper. Med. **42**: 753 (Dec.) 1925.

2. Wilson, J. R., and DuBois, R. O.: Keratomalacia in Infant, with Postmortem Examination, Am. J. Dis. Child. **26**: 431 (Nov.) 1923.

may cause active overgrowth and change in the character of body cells is an important episode, with stimulating possibilities for future developments.

THE CAPILLARY PERMEABILITY OF DIFFERENT ORGANS

Whether the exchange between the blood and the fluid in the lymphatic system proceeds entirely according to simple physicochemical principles was the subject of active discussion thirty years ago. There was an outspoken tendency at that period to abandon all so-called vitalistic explanations of circulatory phenomena in the body so far as current knowledge would permit. Mechanical theories became dominant. In connection with the origin of lymph,[1] it was pointed out that the forces in activity are, first, intracapillary pressure, tending to filter the plasma through the endothelial cells composing the walls of the capillaries; and, second, molecular energy in the form that gives rise to the phenomena of diffusion and osmotic pressure. By the action of this force, the flow of water from one place to another is influenced in accordance with the difference in concentration of the dissolved substances. These two forces acting everywhere control primarily the amount and composition of the lymph.

The foregoing theory, which has enjoyed a long survival of popularity, has not persisted without meeting serious obstacles. There are admitted phenomena that are difficult of explanation on the basis just propounded. The flow of lymph varies widely in different regions of the body, and there are some differences of composition. Consequently there has been a disposition to assume an added physiologic factor—something related to the boundaries between the blood and lymph streams, the cellular vessel walls. A recent writer,[2] in discussing the dilemma, points out that, desirable as it is to draw on physical and chemical forces for the explanation of fluid exchange, it is nevertheless impossible to escape the belief that the individual endothelial cells have the ability to regulate to some degree the transport of substances through their cell wall and cytoplasm. As an example, he adds that the lymph has frequently been considered a product of endothelial activity.

Under the test of experiment, Lee [2] has found anew that a definite change in the blood, such as the introduction of foreign substances, may produce unlike lymphatic responses in different regions. The lymph or tissue fluid derived from the brain capillaries does not invariably correspond with the lymph originating from the capillaries of the viscera under such circumstances. In other words, there seems to be a fundamental difference in the behavior of blood capillaries of different organs. In Lee's tests, the small vessels of the brain did not dilate like the visceral capillaries after histamin was given. They did not indicate the same fluid exchange with crystalloids as did the capillaries in organs drained by the lymphatic system. Furthermore, the passage of particles of India ink from the blood to the lymph vessels of the liver indicated the high degree of permeability in the blood vascular bed of that organ. Accordingly, there is something cogent in Lee's deduction, from such physiologic differences, that the capillaries in every organ are adapted particularly for the function of the organ in question. Whether this adaptation is an inherent quality of the capillary endothelium or whether it is due to influences arising in the tissue around the vessel, or both, cannot be stated. It is obvious, Lee concludes, that explanations based, even though only partly, on known physical and chemical laws are preferable to views depending on indefinite properties of cytoplasm; unfortunately, these laws do not completely suffice, and therefore an active participation of the endothelial cells must be considered.

1. This statement of the current theories is taken from Howell, W. H.: Text-Book of Physiology, Ed. 9, Philadelphia, W. B. Saunders Company, 1925.
2. Lee, F. C.: A Note on Differences of Capillary Activity, Am. J. Physiol. 74: 326 (Oct.) 1925.

UREA AS A DIURETIC

The once widely fostered belief that the intoxication of uremia is due chiefly to substances that are normally eliminated in the urine has practically been abandoned. If any chemical compound is to be held responsible, it seems more likely to be found among more harmful by-products of metabolism that accumulate in the blood. At any rate urea, the most prominent and abundant of all the ordinary urine components, has lost the reputation for pronounced toxicity with which it was formerly endowed. The body will withstand introduction of large quantities of urea without appreciable harm, and this nitrogenous catabolite is freely distributed throughout the fluids and tissues of the organism and appears to be eliminated with comparative ease. According to Hewlett [1] and his co-workers, after the ingestion of 100 gm. of urea by normal persons, the urea nitrogen of the blood can be raised to a figure comparable with that obtained in cases of chronic nephritis. When the blood urea exceeds 70 mg. per hundred cubic centimeters, symptoms of headache, dizziness and slight somnolence may occur. Whether these are due to the sudden alteration in the composition of the body fluids and tissues, or whether they are toxic effects produced by the urea itself, has not been determined.

The foregoing instances represent extremes of urea concentration in the blood. Confidence in the relative harmlessness of urea when the function of the kidneys is good rather than impaired was exhibited more

1. Hewlett, A. W.; Gilbert, Q. O., and Wickett, A. D.: The Toxic Effects of Urea on Normal Individuals, Arch. Int. Med. 18: 636 (Nov.) 1916.

than a quarter century ago when the use of the substance as a diuretic was proposed. That it can be given with impunity to patients over long periods has been demonstrated anew by Crawford and McIntosh [2] at the Hospital of the Rockefeller Institute for Medical Research. They have administered it in doses of from 30 to 60 gm. a day in cases of advanced cardiac decompensation that have not responded satisfactorily to the methods usually employed in the treatment of cardiac edema.

The choice of a diuretic is rarely an easy problem. Urea is rapidly absorbed, and an increase immediately occurs in the height of the urea in the blood. The mean level of the urea in the blood is dependent on the dosage, and the relationship between them is fairly constant in any particular case. The amount of urea excreted depends on the blood urea, so that with constant urea administration a state of equilibrium is reached between the intake and the output. Crawford and McIntosh further point out that during urea diuresis the excretion of water runs more or less parallel with that of urea, so that urine volume reflects the concentration of urea in the blood. Undoubtedly, they add, the explanation of the diuresis is that the excess of urea circulating in the blood is excreted by the kidney, and during the process carries with it a considerable amount of water. With continuous administration, the daily urine volume was maintained at an almost constant level in their patients. The response after administration was rapid, but the effect passed off in a short time, unless the dose was repeated. In contrast with various renal vasodilator drugs and kidney cell irritants, urea seems to function well in promoting a normal water balance in the body. The New York clinicians believe that it deserves a greater popularity.

Current Comment

IS THE PITUITARY ESSENTIAL TO LIFE?

Information regarding the actual functions of the pituitary gland is singularly conflicting. In few domains of physiology are equally contradictory data supplied by investigators of scientific repute.[3] Contrary to the earlier students of the subject, Paulesco [4] maintained the thesis that the hypophysis is necessary to life. His conclusions were based on the outcome of surgical extirpation of the pituitary and were subsequently supported by the independent observations of a number of experienced investigators, including Cushing and his co-workers in this country. It might be assumed that the failures to produce fatal conditions through hypophysectomy have been due to incomplete operations; yet postmortem examinations have left no warrant for such a conclusion. An explanation of the divergent results appears to have been furnished by Dandy and Reichert [5] of the department of surgery at the Johns Hopkins Hospital. They point out that, as an organ well secluded at the base of the brain and skull and attached to both at the central point, the hypophysis has offered great difficulties of operative approach preparatory to its extirpation. It seems likely, therefore, that for the seemingly paradoxical results now on record the operative procedure itself must be held responsible. Cerebral trauma is not easily averted. Lassitude, drowsiness and even coma, as well as convulsions, are occasional complications of human intracranial operations; and it has been precisely such symptoms that have attended the experimental hypophysectomies which have had a fatal outcome. Dandy and Reichert have perfected an operative technic that permits the safe removal of the hypophysis in dogs, the species about which the controversies have revolved. With their procedure the Baltimore surgeons have, as they express it, found no evidence to support, and every evidence to refute, the assumption that the hypophysis is essential to life, or that the group of symptoms described by others as preceding death in hypophysectomized animals is of hypophysial origin.

BACTERIOPHAGE NEWS

There is a fascinating aspect to the conception that bacteria are subject to enemies within their own camp. Mankind has been industrious in devising ways of destroying the harmful micro-organism by a great variety of devices. Chemical as well as physical agents have been requisitioned in this bitter warfare; and when there has been no adequate kind of ammunition devised or available, defensive barriers have been erected. Antisepsis has vied with asepsis as a protective device. There can be no cessation of activities, for the menace is ever present and seizes every advantage of laxity on the part of the armies of preventive medicine. Lack of preparedness invites momentary human disaster. In the midst of this tension has come the news of the bacteriophage, the reputed discovery of a filtrable, ultramicroscopic virus, parasitizing on and destroying actively growing, susceptible bacteria. Such a prospect of unsuspected potent help has been hailed with interest and enthusiasm not only by the scientist but also by the layman. Sinclair Lewis gave it publicity as a leading theme in the much discussed novel "Arrowsmith." It was d'Herelle's idea that there is only a single bacteriophage, common to man and animals, capable by adaptation of acquiring a virulence toward all bacterial species.[6] This view of the unity of the lytic principle has been considerably debated. Obviously the determination of whether there is a multiplicity of bacteriophagic agents, each of which is more or less specific for definite microbes, is of great importance in attempts to employ the

2. Crawford, J. H., and McIntosh, J. F.: The Use of Urea as a Diuretic in Advanced Heart Failure, Arch. Int. Med. **36**: 530 (Oct.) 1925.

3. The Pituitary Gland, Glandular Therapy, Chicago, American Medical Association, 1925, p. 50. Howell, W. H.: Physiology of the Pituitary Gland, J. A. M. A. **83**: 1684 (Nov. 22) 1924.

4. Paulesco, N. C.: L'hypophyse du cerveau, Paris, Vigot Frères, Eds., 1908.

5. Dandy, W. E., and Reichert, F. L.: Studies on Experimental Hypophysectomy, I, Effect on the Maintenance of Life, Bull. Johns Hopkins Hosp. **37**: 1 (July) 1925.

6. D'Herelle, F.: The Bacteriophage, English Translation by G. H. Smith, Baltimore, 1922.

bacteriophage in the warfare against infection. At the Rockefeller Institute for Medical Research, Bronfenbrenner and Korb[7] have lately observed persistent differences in the individual resistance of lytic filtrates active against different micro-organisms in ways that lead them to favor the conception of the multiplicity of bacteriophages. This dissipates, for the moment, at least, the hope of a single universal panacea for those infected with bacteria.

Association News

THE DALLAS SESSION

Special Exhibit by the Section on Dermatology and Syphilology

In connection with the Dallas Session, the Section on Dermatology and Syphilology will again conduct an exhibit. The committee in charge is composed of E. D. Crutchfield, Galveston, Texas; Fred D. Weidman, Philadelphia, and I. L. McGlasson, San Antonio, Texas, chairman. According to the plans of the committee, the exhibit will not be miscellaneous but planned to illustrate the various phases of cancer, in all stages. It is intended to make it of interest to the profession at large as well as to the members of the section.

Circular letters explaining the exhibit, with application blanks, will be mailed to those who registered in the Section on Dermatology and Syphilology during any of the last five sessions. Others who desire this literature may obtain it by communicating with I. L. McGlasson, City National Bank Building, San Antonio, Texas. All applications must be sent to Dr. McGlasson by February 1.

Medical News

(PHYSICIANS WILL CONFER A FAVOR BY SENDING FOR THIS DEPARTMENT ITEMS OF NEWS OF MORE OR LESS GENERAL INTEREST: SUCH AS RELATE TO SOCIETY ACTIVITIES, NEW HOSPITALS, EDUCATION, PUBLIC HEALTH, ETC.)

CALIFORNIA

Personal.—Dr. Randolph F. Hunter and Dr. Harold W. Comfort have taken up their duties as chief surgeon and associate physician, respectively, at the Scotia Hospital, Scotia, succeeding Drs. Charles C. Cottrell and Frank K. Haight, both of whom recently resigned to engage in private practice.

Resolution About Anesthetists.—The Community Hospital, Long Beach, recently passed the following resolution regarding anesthetists:

WHEREAS, The California Medical Association has declared that the administration of anesthesia is the practice of medicine.

WHEREAS, The attorney-general of the state of California has ruled that the giving of an anesthetic is the practice of medicine.

WHEREAS, The board of medical examiners of the state of California has ruled that the giving of an anesthetic is the practice of medicine.

WHEREAS, It is against the spirit of the law, against the ethics of medicine, and against the ruling of a large majority of accredited hospitals for nurses or any one else not legally qualified to practice medicine to give an anesthetic in California.

WHEREAS, The superior courts of two states of the United States have already handed down decisions fixing the liability for damages against hospitals in which nurses have administered anesthetics at the time of the death of the patient while under influence of the anesthetic, judgments against the hospital to the extent of $10,000 for damages in one case, and $150,000 in another case having been obtained.

WHEREAS, The Community Hospital of Long Beach intends to comply with the spirit of the laws of the state of California, the code of ethics of the American Medical Association, and all the requirements for an accredited hospital; therefore, be it

Resolved, That the administration of general and local anesthesias in the Community Hospital of Long Beach be limited and restricted exclusively to Doctors of Medicine (M.D.'s) legally qualified to practice medicine and surgery in the state of California.

7. Bronfenbrenner, J. J., and Korb, C.: Studies on the Bacteriophage of d'Herelle. IV. Concerning the Oneness of the Bacteriophage, J. Exper. Med. 42:821 (Dec.) 1925.

FLORIDA

En Route to Florida.—The secretary of the California State Board of Medical Examiners notified the Florida Board of Examiners, November 10, that one A. L. Oxford, who had in some way obtained the California license to practice medicine of Dr. Fletcher A. Butler, deceased, was on his way to Florida. A complaint was filed against Oxford in Los Angeles, November 7, charging violation of the medical practice act; he is also said to have been arrested in 1924 for petty larceny.

GEORGIA

Pleadings in Suit for Fees.—The Georgia Court of Appeals has affirmed a judgment by which Dr. D. Granville Humphreys of Fernandina, Fla., was awarded $310, against J. H. Becker of St. Mary's, Georgia, for services rendered a third person at Becker's request. Liability was denied by Becker in the court of appeals because Humphreys had not alleged in his pleadings that he had been licensed to practice in Georgia. The court held that in an action for the value of professional services, it is sufficient for a physician to allege that he is a physician, that he was employed as such, that he rendered the services alleged, and that the reasonable value of such services was the amount sued for. The court held further that the law does not raise an implied promise by a third person to pay for professional services rendered a patient at his request, but that a physician must prove an understanding between himself and the third person that the third person, and not the patient, would pay.

IDAHO

Personal.—The governor has appointed Dr. Ralph M. Fouch, American Falls, medical adviser of the state department of public welfare, to succeed Dr. Frank W. Almond, resigned.

ILLINOIS

Scarlet Fever Increasing.—The prevalence of scarlet fever in the state is 25 per cent. in excess of what it was this time last year, and, according to the state department of health, it is steadily increasing; 414 cases were reported last week, whereas 109 were reported the first week in October. The department says that the greatest monthly prevalence of scarlet fever in the state habitually occurs in January.

Chicago

Medical History Club.—The medical history club, recently organized at the University of Illinois College of Medicine, was addressed by Mr. Christian Bay of the John Crerar Library, November 4, on "Observations of a Medical Librarian Abroad"; by Arthur H. Daniels, Ph.D., dean of the Graduate School, University of Illinois, December 2, on "The Philosophy of Spinoza," and by Dr. Morris Fishbein, December 16, on "Eclecticism." The next lecture will be by Dr. Basil C. H. Harvey of the University of Chicago, on "Vesalius."

Dr. Falls Appointed Professor at Illinois.—Dr. Frederick H. Falls, Iowa City, has accepted the appointment of professor and head of the department of obstetrics and gynecology at the University of Illinois College of Medicine, Chicago. Dr. Falls, who formerly was an instructor in that department at Illinois, has since 1921 been head of the department of obstetrics and gynecology at the State University of Iowa College of Medicine, Iowa City; he is a graduate of the University of Chicago and Rush Medical College, and has a master's degree from the University of Illinois. ——The announcement in THE JOURNAL, December 12, of the appointment of Dr. Carl A. Hedblom should have been as professor and head of the department of surgery at the University of Illinois.

Rawson Laboratories Dedicated.—The new Rawson Laboratories connected with Rush Medical College were dedicated with appropriate ceremonies, December 18. The building is five stories high, of steel construction, joined on one side with the Presbyterian Hospital and on the other with the Senn Memorial building. On the first floor are administrative offices and library built for more than 25,000 volumes; the second, third and fourth floors are for classrooms, examining rooms, reception rooms and special laboratories. Surgery occupies the entire fourth floor, and the Norman Bridge pathologic laboratories the fifth floor. The Central Free Dispensary, which last year treated more than 100,000 persons, will be in this building and the Senn Memorial building. There is a children's play room for mothers who, in seeking treatment, bring their children, and a play director

in charge. There are small treatment rooms throughout the building, two operating rooms on the fourth floor for minor operations, and various small laboratories for medical research for advanced students. The laboratories have the latest improvements. The building generally is of the most modern type.

INDIANA

New Indianapolis Board of Health.—According to the Indianapolis *News*, Mayor-elect Duvall has announced the selection of Dr. Everett E. Padgett to be president of the board of health, and of Dr. William E. Mendenhall, Dr. Arthur E. Guedel and Dr. Frederick E. Jackson as members.

Personal.—Dr. Charles L. Cabalzer, Indianapolis, has been elected president of the staff of the Methodist Hospital for the coming year.——Dr. William A. Buchanan has been appointed for the third time commissioner of health of Hammond.——Dr. Uryal C. Ambrose has been appointed a member of the board of directors of the Indiana Christian Hospital, Indianapolis.——Mayor-elect Tinkham of Gary has announced the appointment of Dr. William M. Bigger as president of the board of health, and Dr. William A. Buchanan as secretary.

Annual Report of State Board of Health.—The birth rate in Indiana in 1924 was the highest recorded in ten years (22.4); the death rate from diphtheria, scarlet fever, tuberculosis and maternal causes, and the infant mortality rate were the lowest on record. There were 534 deaths in the state from automobile accidents, as against 362 in 1922, and this cause now leads all other accidental causes of deaths. Among 208 brains of dogs and other domestic animals examined for rabies, seventy-six were positive, and ninety-two persons were given the Pasteur antirabic treatment in the laboratory. The division of school hygiene made sanitary surveys of 139 school buildings, condemning thirty-eight, practically all of which were old one-room structures; the division also approved plans for ninety-three new school buildings and specifications for remodeling seventy-seven old school buildings. Health films were shown to more than 23,000 people by the division of child hygiene, which also held 1,938 mothers' classes, with an attendance of about 48,000.

KANSAS

Personal.—Dr. Fred C. Cave, Grainfield, who has been appointed full-time health officer for Ellis County, assumed his duties, November 1.——Dr. Sydney P. Loomis, formerly of Lost Springs, has purchased the Virginia Hotel at Herington, which he will convert into a hospital.

LOUISIANA

Hospital News.—The French Hospital, New Orleans, which for sixty years has been maintained by a society of persons of French descent, is being remodeled from a forty-five bed hospital to a ninety-bed hospital and will be at the service of the entire city. The institution is owned by the Société Française de Bienfaisance et Assistance Mutuelle.

Gifts to Hospitals.—Mr. Luca Vaccaro has given $10,000 to the Southern Baptist Hospital, New Orleans, to equip the roentgen-ray department. The hospital will be formally opened in January.——Mrs. John Dibert has given to the Eye, Ear, Nose and Throat Hospital, New Orleans, another gift of $50,000, making her total donation to that institution $135,000.

Society News.—The Orleans Parish Medical Society, New Orleans, has unanimously recommended Mr. F. W. Evans, vice chairman of the board of Charity Hospital, for the award of the annual *Times-Picayune* cup given to the citizen doing the greatest work of outstanding character.——The annual dinner of the Orleans Parish Medical Society was held, December 12, following the annual election.

Professor Castellani En Route to New Orleans.—Prof. Aldo Castellani sailed from London, December 16, for New York en route to New Orleans to take up his duties as professor and head of the department of tropical diseases at Tulane University of Louisiana School of Medicine. According to Dean Bass, new office and laboratory equipment is now being installed in the Hutchinson Memorial building so that Professor Castellani can take up the organization of his department on arrival. It will be essentially a research department. It will provide service for the Charity Hospital, and give instruction to the undergraduate classes. Although not formally enrolled in the faculty of the graduate school, Professor Castellani will give graduate instruction (THE JOURNAL, November 7, p. 1494).

MARYLAND

Joint Meeting on Cancer.—A joint meeting of the Maryland Cancer Committee, the Baltimore City Medical Society and the Baltimore City Dental Society was held, December 18, at Osler Hall, Baltimore, to discuss "Research Work on Cancer in This Country and Abroad." Dr. George A. Soper, managing director, American Society for the Control of Cancer, who has just returned from Europe, discussed the work of Gye and Barnard; Dr. William H. Woglom, associate professor of cancer research, Columbia University College of Physicians and Surgeons, New York, discussed the experimental side of cancer in relation to diagnosis and treatment, and Dr. William H. Welch, director, Johns Hopkins School of Hygiene and Public Health, discussed both subjects.

Anniversary of Medical School.—The one hundred and eighteenth anniversary of the founding of the School of Medicine of the University of Maryland was commemorated, December 17, by a banquet at the Hotel Emerson, which members of the faculty and alumni from New York, Philadelphia, Baltimore, Washington and other cities attended. Chartered by the legislature, Dec. 7, 1807, as the College of Medicine of Maryland, it was the first unit of the University of Maryland, the sixth institution of collegiate standing to be founded in Maryland, and the third in Baltimore, and also the fifth medical school to be established in this country. Dr. John B. Davidge, who settled in Baltimore in 1796, the year in which the city received its charter, was the founder, and Dr. James Cocke, a native of Virginia, and Dr. John Shaw of Annapolis were his associates. At the banquet, December 17, Dr. Arthur M. Shipley, professor of surgery, was toastmaster, and among the speakers were Dr. William S. Love, president, alumni association; Dr. Maurice C. Pincoffs, professor of medicine, and Dr. James M. H. Rowland, dean of the medical department.

MASSACHUSETTS

Personal.—George E. Vincent, Ph.D., president of the Rockefeller Foundation, New York, will deliver an address at the celebration of the fiftieth anniversary of the Boston Medical Library, Jan. 19, 1926.

Hospital News.—The Florence Crittenden League of Compassion has dedicated a new maternity hospital and home on the site of the old Peter Faneuil homestead, Oak Square, Brighton. The league is conducting a campaign to raise $150,000 to carry on this work.

Society News.—Dr. Harry C. Solomon, Boston, addressed the North Shore Medical Fraternity at Lynn, November 24, on "Epilepsy: Its Various Forms and Treatment."——Prof. Vittorio Putti addressed the Harvard Medical Society at the Peter Bent Brigham Hospital, Boston, November 30, on "History of the University of Bologna."

MICHIGAN

Diphtheria Immunization at Niles.—Local physicians and nurses cooperated with the health officer of Niles during the last four weeks to administer toxin-antitoxin to the 1,200 school children to immunize them against diphtheria. According to the Niles *Star*, this work was completed before there had been any serious outbreak of diphtheria this year. This was part of the program of the state board of health to eradicate diphtheria from the state. The physicians donated their services for this work.

Society News.—Dr. John Shelton Horsley, Richmond, Va., addressed the Wayne County Medical Society, Detroit, December 21, on "Biologic Principles Underlying Gastric Surgery."——Dr. Herman B. Van Wyck of the University of Toronto Faculty of Medicine addressed the Bay County Medical Society at Bay City, recently, on "Toxemias of Pregnancy."——The Alpena County Medical Society put on a program at Bay City recently in return for a program presented by local members at Alpena last May.——Dr. Hugh T. Patrick, Chicago, emeritus professor of neurology, Northwestern University Medical School, addressed the annual meeting of the Kent County Medical Society, Grand Rapids, December 16, on "Some Phases of Psychanalysis."

MINNESOTA

Society News.—Dr. David L. Tilderquist has been elected president of the Minnesota Academy of Ophthalmology and Otolaryngology, and Dr. John H. Morse, secretary-treasurer.

Outbreak of Poliomyelitis.—At the annual meeting of the Minnesota State Sanitary Convention, St. Paul, November 5-6, Dr. Orianna McDaniel, director of the division of pre-

ventable diseases, state department of health, stated that there had been ninety-one deaths so far in the outbreak of poliomyelitis in the state since June 1, and that cases had been reported from eighty-one of the eighty-seven counties.

MISSOURI

The Adcox Confession (continued).—Adcox compares the "opening up" of Connecticut to "bringing in" a gusher in an oil field. He says in the St. Louis *Star*, that he knew nothing about conditions in Connecticut, heretofore, but he decided to tap the field with two unusually dumb men, who had $1,500 each to pay for a license. Adcox attempted in other places to get licenses for these boys, but they were so stupid that both failed to get by after he had things fixed. He called them to his home and offered to take them to Connecticut, provided they paid expenses. On arrival at the Guarde Hotel, New Haven, Adcox warned his "dumb-bells" to stay in their rooms and not talk to anybody, then went to the lobby and met the "famous fixer" who had opened up Arkansas. "Famous fixer" forbade Adcox to work in Connecticut, but the next morning, Adcox and his "dumb-bells" were the first to arrive at the examination room. Adcox, sitting between the two, wrote the examination himself and passed answers, fore and aft, for practically all the questions. "Famous fixer" and his delegation obtained the questions to be asked the next day and worked all night writing up their papers. All but one of "famous fixer's" clients passed the examination, and he was so illiterate that the board could not read his papers. Adcox's "dumb-bells" passed, but he failed, having been so busy helping others and finding out what was going on. Before the party broke up, "famous fixer" warned Adcox that although he got by this time in Connecticut, he had seen to it that he would not be on deck the next trip; he told the truth, for shortly after Adcox returned to St. Louis, "we were informed" that the diplomas of the St. Louis school would no longer be recognized in Connecticut. Adcox had at that time fifteen prospects who wanted to make the next trip. The author takes time out here to say that in all the history of medicine or of state government there has never been anything as rotten as the scandal in Connecticut. There were no requirements or restrictions, and any person willing to pay could obtain a license. The type of person that took advantage of the offer was the ignorant immigrant who knew little English and the lowest of laborers; men who had driven dump carts in January were administering anesthetics and operating for appendicitis in February; scores of murders were committed in the name of science, he says, and were covered up by phraseology in death certificates. Men too ignorant to be called quacks, who would not be tolerated in any other state, found a haven in Connecticut, and they organized, and became a potent political power. It was this, Adcox says, which he saw with his own eyes, that brought him to realize the seriousness of his offenses and caused him to determine to end his career; not yet, however, for he made plans to defeat the "famous fixer" by sending him prospects who would purchase diplomas from his institution. With Connecticut refusing now to admit applicants from the St. Louis school, Adcox sent his "graduates" to Kansas City to graduate again. "Famous fixer" not only controlled the situation in Connecticut but actually set the dates for the state board examinations; he brought with him to the Guarde Hotel, New Haven, blank diplomas for the easterners who had not gone west for "commencement" and for such unexpected applicants as put in an appearance. He failed once to obtain the questions in advance, so he called his "applicants" together and told them to go slow and he would get the questions at noon. He not only kept his word but had the meeting of the board adjourned until the next morning and devoted the afternoon and night to preparing examination papers.

Ralph Voigt went to Connecticut on one trip for Adcox, taking several "young men." He arrived too late for the examination, but a member of the board took the whole delegation to a hotel and gave oral examinations; one of this bunch, Adcox says, now has a large practice in Richmond as an eye specialist. On his numerous visits, Adcox saw matters steadily growing worse in Connecticut. Hospitals were being operated by quacks, every known "ism" was practiced, and quacks were selling their whisky prescriptions for $3 apiece. He saw now that the diploma mill had grown too big to control and that it must soon fall by its own weight. He returned to St. Louis from Connecticut, and resolved to quit the business. Before long, however, a young man who had been his neighbor for several weeks came to his home with his throat swathed in bandages. While Adcox was treating him for tonsillitis, he indicated that he had always wanted to be a doctor but had never had the chance; here, then, was another prospect, so Adcox offered to make "Harry Thompson" a doctor.

NEW JERSEY

Hospital News.—The new $1,000,000 Englewood Hospital, paid for wholly by popular subscription, was dedicated, November 27; officials and residents of twenty-two communities in New Jersey's Northern Valley attended the ceremony.

Society News.—Dr. Arthur C. Morgan, Philadelphia, conducted a bedside clinic before the Atlantic County Medical Society, November 15, at the City Hospital, and addressed the society on "Internal Medicine."——Dr. Livingston Farrand, president of Cornell University, Ithaca, N. Y., addressed the Essex County Medical Society at Newark, December 10, on "The Responsibilities of the Medical Profession to the Community."

NEW YORK CITY

Dr. Wood to Investigate Cancer Research.—Dr. Francis Carter Wood, vice president of the American Society for the Control of Cancer, sailed, December 9, according to press reports, for Europe to investigate the various recent reports on cancer research in England, and on his return will make an official report of his investigation.

Medical Diploma Stolen.—Dr. James J. Short, 110 West Fifty-Fifth Street, writes that a news item in THE JOURNAL, November 7, prompted him to report that about a year ago his medical diploma disappeared as the result of a robbery which took place while he was making professional calls. Dr. Short graduated from the University of Buffalo Department of Medicine in 1918.

Another Convalescent Home.—The family of the late J. Kennedy Tod has given his estate at Oyster Bay to the Columbia-Presbyterian Medical Center for a convalescent home for patients. The property includes two small islands and a peninsula of 117 acres extending into Long Island Sound; also permanent buildings and a long sandy beach suitable for salt water bathing.

Society News.—Dr. Wendell C. Phillips, president-elect of the American Medical Association, addressed the First District Dental Society at the Hotel Pennsylvania, December 3. ——Dr. J. L. Amster addressed the Bronx Surgical Society, November 25, on "Postoperative Fecal Fistual Communicating with Ovarian Dermoid," and Dr. Benjamin Sherwin on "The Treatment of Acute Peritonitis."——Dr. Philip Liebling addressed the Bronx Gynecological and Obstetrical Society, November 30, on "Purpura Hemorrhagica."——The twentieth annual meeting of the Woman's Medical Society at New York will be held at the Hotel McAlpin, March 29, 1926.——Dr. S. Adolphus Knopf addressed the Medical Association of the Greater City of New York, December 21, at the New York Academy of Medicine on "Birth Control; Sanitary, Medical and Moral Aspects."——Dr. Thomas J. Harris addressed the New York Academy of Medicine in a program arranged in cooperation with the Section of Laryngology and Rhinology, December 17, on "End-Results of Tonsillectomy, with Especial Reference to the Legal Responsibility."——The Riverside Medical Society gave a dinner, December 15, at the Hotel Majestic in honor of Dr. Lewis G. Cole, who addressed the society on "Gastric Motor Phenomena," illustrated with motion pictures.——Dr. Emilius C. Dudley, emeritus professor of gynecology, Northwestern University School of Medicine, Chicago, addressed the Bay Ridge Medical Society, December 8.——Dr. Edward A. Strecker, Philadelphia, addressed the Medical Society of the County of New York, December 28, on "The Psychoneuroses with Particular Reference to Their Management."——The Yorkville Medical Society conducted a symposium on "Blindness Due to Sinus Disease," December 21, arranged by the otolaryngological department of Beth Israel Hospital, and were addressed by Dr. Ralph Almour on "The Ear as a Diagnostic Aid in Syphilis," and by Dr. Mervin C. Myerson on "Medical Bronchoscopy."

OHIO

Dr. Lavender Appointed Health Officer.—Dr. De Witt C. Lavender has been appointed health commissioner of Mansfield and Richland County to succeed Dr. William De Kleine, resigned, who will go to Fargo, N. D., to take charge of the American Child Demonstration there. Dr. Lavender heretofore was the deputy health commissioner.

Course in Plastic Surgery.—For the first time in this country since the World War, a course in plastic facial surgery

was given this month at the University of Cincinnati College of Medicine, under the direction of the departments of anatomy and otolaryngology; the instructors were Dr. Oscar V. Batson, professor of anatomy, and Dr. Samuel Iglauer, professor of laryngology, assisted by Dr. Jacques Maliniak.

Professor Mathews on Leave.—Albert P. Mathews, Ph.D., professor of biochemistry, University of Cincinnati College of Medicine, Cincinnati, is in England on sabbatical leave, working with Prof. Edward C. Baly of the department of physical chemistry, and with Professor Ramsden of the department of physiology, at the University of Liverpool; he will also be at Oxford a short time to visit Professor Halliburton in London, and spend several months on the continent, and next June he will attend the celebration of the five hundredth anniversary of the University of Louvain.

Personal.—Dr. John H. Baird, formerly with the Veterans' Hospital in the Bronx, New York, has been detailed as clinical director of the Veterans' Bureau Hospital No. 97, at Camp Sherman, near Chillicothe.——Dr. Perry I. Tussing was elected president of the Allen County Academy of Medicine, Lima, November 17, at which time he addressed the academy on "Diseases of the Heart."——Dr. Robert C. Longfellow, Toledo, is at present house physician for American and English-speaking tourists at the Khedivial Hotel, Cairo, Egypt; he will return next spring.——Dr. M. Murray Levine has resigned from the staff of the Cleveland State Hospital, Cleveland, to engage in private practice in that city.——Dr. Trent A. B. Denison has resigned as superintendent of Lakeside Hospital, Cleveland, to engage in private practice.——Dr. William J. M. Scott, instructor in surgery, Western Reserve University School of Medicine, Cleveland, is leaving to accept the position of assistant professor of surgery in the new Rochester Medical School, Rochester, N. Y., Jan. 1, 1926.——Dr. Emile Holman, assistant professor of surgery, Western Reserve University School of Medicine, is leaving to accept the position of associate professor of surgery at Stanford University, California, Jan. 1, 1926.——Dr. James M. Snider, Marysville, has been appointed a member of the Union County Board of Pension Examiners, succeeding the late Dr. Lutrelle Henderson.——Dr. John DeForest Smith has been reelected president of the Horticultural Society of Dayton.——Dr. Ralph W. Holmes, Chillicothe, has been elected president of the Ross County Academy of Medicine.——Dr. Aaron F. Basinger has been elected president of the medical staff of the City Hospital, Lima.

OREGON

License Revoked.—The state board of medical examiners reports that the reciprocity license of Dr. Wilbur W. Illsley, Hermiston, Ore., was revoked at a meeting of the board, November 12; Dr. Illsley is still licensed to practice osteopathy in Oregon.

PENNSYLVANIA

Society News.—Dr. William D. Stroud of the Graduate School of Medicine, University of Pennsylvania, Philadelphia, addressed the Berks County Medical Society at the November meeting on "Prognosis in Heart Disease."——Dr. Alexander H. Colwell addressed the Fayette County Medical Society at Union Town, November 5, on "The Treatment of Emergencies in Heart Practice."——Dr. Thomas G. Simonton, associate professor of medicine, University of Pittsburgh School of Medicine, and Dr. Harold A. Miller, professor of obstetrics, University of Pittsburgh School of Medicine, addressed the Mercer County Medical Society recently on "Referred Pain in Pneumonia" and "Pyelitis in Pregnancy," respectively.

Pittsburgh Skin and Cancer Foundation.—In the annual report of this foundation, President Judge Reed shows that the attendance in its second year has doubled; that 557 new cases were received and 301 clinics held; of the new cases 410 were in the new skin department, ninety-three in the cancer department, of which sixteen were referred to private physicians, twenty-two were improved, two died and fifty-three are still under treatment. A department of gastroenterology has been established under the direction of Dr. Herbert L. Jones, and a study is being made to determine any relation that may exist between gastro-intestinal and dermatologic conditions. The treatment rooms and office are at 1901 Fifth Avenue.

Philadelphia

Personal.—Dr. Edmund B. Piper has been appointed on the gynecologic and obstetric staff of the Philadelphia General Hospital to fill the vacancy caused by the death of Dr. John Cooke Hirst.

Society News.—Dr. James F. Donnelly has been elected president of the Philadelphia Clinical Association; Drs. Ernest G. Maier and Samuel P. Gerhard, vice presidents; Dr. William H. Thomas, treasurer, and Dr. Louis R. Wiley, secretary.

Biologic Abstracts.—The Rockefeller Foundation has given $350,000 to establish a biologic abstract service in Philadelphia covering the whole field of pure and applied botany and zoology. *Botanical Abstracts, Abstracts in Bacteriology* and the abstracting service of several other journals will be merged with the new service. According to the *Atlantic Medical Journal*, about 35,000 abstracts will be published annually, including papers prepared through the collaboration of biologists. The service will be conducted under the auspices of the Union of Biologic Societies of America, with headquarters at the University of Pennsylvania. The monthly journal will be known as *Biological Abstracts*. Jacob R. Schramm, Ph.D., of Cornell University, Ithaca, N. Y., has been made editor in chief and John A. Detlefsen, Sc.D., formerly of the University of Illinois and the Wistar Institute of the University of Pennsylvania, associate editor in the field of animal biology.

RHODE ISLAND

Society News.—Dr. Wilfred Pickles addressed the Providence Medical Association recently on "Sacral Anesthesia," and Dr. Elliot A. Shaw on "Some Problems Presented by Patients with Jaundice." Dr. Robert M. Lord addressed the association, November 2, on "A Report on Fifty Cases of Pyelitis in Children."

TENNESSEE

Tri-State Association Meeting.—The forty-second annual meeting of the Tri-State Medical Association will be in Memphis, Jan. 26-28, 1926, at the Hotel Peabody. This organization has held annual meetings ever since its founding in 1883, except in 1917 and 1918; last year the association honored Dr. Henry C. Dunavant of Osceola, Ark., the surviving member of the committee which wrote the constitution in 1883. Five members from each of the three states have been selected to present papers at this meeting in addition to other speakers, among whom will be Dr. Martin H. Fischer, professor of physiology, University of Cincinnati School of Medicine; Dr. Rudolph Matas, professor of general and clinical surgery, Tulane University of Louisiana School of Medicine; Dr. Thomas R. Brown, professor of clinical medicine, Johns Hopkins University Medical Department; Dr. Julius H. Hess, professor and head of the division of pediatrics, University of Illinois College of Medicine, and Dr. Bethuel B. Vincent Lyon, Philadelphia.

TEXAS

Publicity in Common.—The Dallas County Medical Society, Dallas, recently passed the following resolution:

Resolved, That in appreciation of the scientific standards maintained by the dental profession generally, and out of the high regard that the Dallas County Medical Society has for the Dallas Dental Society, we extend an invitation to have such publicity committee as may exist, or may be appointed by their president, meet with a like committee of our own, to discuss publicity in which the two professions have a common interest.

Society News.—Dr. Charles S. Hale, Cisco, has been elected president of the Eastland County Medical Society.——Dr. Ewin P. Hall, Fort Worth, addressed the Tarrant County Medical Society, recently, on "The Management of a Case of Obstetrics in the Home."—Dr. Louis K. Patton, Amarillo, addressed the Panhandle District Medical Society at Clarendon, recently, on "Mercurochrome Intravenously," and Dr. Robert B. Giles, Dallas, on "The Significance of Heart Murmurs."

WASHINGTON

Society News.—Dr. Alfred H. Winkel recently addressed the Seattle Academy of Medicine on "Thoracoplasty."——The King County Medical Society, Seattle, gave a complimentary get-together dinner at the Olympic Hotel, December 14; all members who read a prepared speech and those talking more than ten minutes were fined $25; there will be no meeting of the medical section of the society, December 21, on account of the holidays.

Cult Bills.—The Public Health League of Washington notes in the *Messenger* that the cults have introduced bills in the present legislature which would provide a punishment of five years in prison for any one who gives or permits to be given any narcotic drug to any person under 21 years of age;

another bill would open up the hospitals of the state to chiropractors, sanipractors and other cults, and another would require physicians to write prescriptions in English and tell patients whenever there is a narcotic included.

GENERAL

Society News.—At the eleventh annual meeting of the Radiological Society of North America, Cleveland, December 7-11, Dr. Manly J. Sandborn, Appleton, Wis., was installed as president; Dr. Edward W. Rowe, Lincoln, Neb., made president-elect; Drs. Bertram C. Cushway, Chicago, Wesley H. Wallace, Brooklyn, and Raymond G. Taylor, Los Angeles, vice presidents, and Dr. Robert J. May, 5005 Euclid Avenue, Cleveland, secretary-treasurer.

Health Contest of Women's Clubs.—The General Federation of Women's Clubs is offering a prize to the state which shows the greatest percentage of clubs that participate in its program of health "surveys," by which the federation aims to secure a cooperative study by groups of the health laws and practices in communities. The federation is being aided in this work by the Metropolitan Life Insurance Company, the American Child Health Association and the American Public Health Association.

Resolution on Alcoholic Liquors.—The National Woman's Christian Temperance Union passed a resolution at the convention in Detroit in November to the effect that the best way to discourage the therapeutic use of alcohol and to relieve pharmacists of their present position of having to handle alcoholic liquors to fill prescriptions of physicians would be to make the sale and prescription of alcoholic beverages as medicine unlawful. The resolution commends the legislature of those states which have passed laws forbidding the sale and prescriptions of alcoholic beverages as medicine.

National Board of Medical Examiners.—There will be examinations held by the board in Parts I and II, February 10-12, in all Class A medical schools desirous of holding the examination and having a sufficient number of candidates, except in large cities, where they will be held in only one school. Candidates should notify the board's office, 1600 Walnut Street, Philadelphia, promptly; those not registered with the board sending formal applications not later than January 15. Examinations in Part III have been scheduled for Chicago in December; St. Louis, December 14-17, and New York City, Jan. 4-7, 1926.

Tax Reduction Bill Passes House.—With an amendment to reduce to the prewar rate the annual tax on physicians under the Harrison Narcotic Law, the federal tax reduction bill passed the House of Representatives, December 19. The amendment fixes the annual license fee of physicians under the narcotic law at one dollar instead of three, which has been the fee since the war period. The amendment was accepted unanimously in the House, and it is expected that the Senate will promptly accept it. Although the Secretary of the Treasury has never affirmatively approved of this tax reduction, it is known that the treasury department will not interpose objections to this amendment. The program of the administration leaders is to have the entire tax legislation disposed of in the Senate by March 1, 1926, so that the reductions will be effective March 15, when income tax returns must be filed. Since the House Ways and Means Committee held hearings on all the essential portions of the measure, the Senate Committee on Finance plans not to hold hearings on the tax bill, as such hearings would delay action. The reasons for the reduction of the narcotic tax on physicians were fully presented to the House Ways and Means Committee by Dr. Charles W. Richardson, Washington, D. C., and Dr. William C. Woodward, Chicago, both representing the American Medical Association. No objection to the adoption of the reduction has thus far appeared from any source.

New County Society Presidents.—The following physicians have recently been elected president of the county medical society indicated:

Dr. Alfred R. Autrey, Port Arthur, Texas, Jefferson County Medical Society.
Dr. Francis B. Blackmar, Columbus, Ga., Muscogee County Medical Society.
Dr. Edwin G. Bloor, Antigo, Wis., Langlade County Medical Society.
Dr. Robert K. Buford, Charleston, W. Va., Kanawha County Medical Society.
Dr. Ebenezer P. Clapper, Waynoka, Okla., Woods County Medical Society.
Dr. Lawrence G. Clayton, Central, S. C., Pickens County Medical Society (reelected).
Dr. William M. Gambrell, Belton, Texas, Belle County Medical Society.
Dr. Edward W. Gray, Sheffield, Ala., Colbert County Medical Society.
Dr. John F. Harrison, Mexico, Mo., Audrain County Medical Society.
Dr. O. Paul Humpstone, Brooklyn, Medical Society of the County of Kings.
Dr. Oren M. Kramer, Millersport, Ohio, Fairfield County Medical Society.
Dr. Bruce D. Lung, Kokomo, Ind., Howard County Medical Society.
Dr. William C. Moser, Morgantown, W. Va., Monongalia County Medical Society.
Dr. William J. B. Orr, Smithfield, N. C., Johnston County Medical Society.
Dr. William G. Pittman, Pine Bluff, Ark., Jefferson County Medical Society.
Dr. Theodore J. Redelings, Marinette, Wis., Marinette-Florence County Medical Society.
Dr. Charles I. Shaffer, Ralphton, Pa., Somerset County Medical Society.
Dr. Lawrence Shields, Xenia, Ohio, Greene County Medical Society.
Dr. Henry M. Stuckey, Sumter, S. C., Sumter County Medical Society.
Dr. Frederick J. Swift, Maquoketa, Iowa, Jackson County Medical Society.

Epidemiologic Report of League of Nations.—The November 15 issue of this report shows a marked increase of plague in August and September in certain regions of southern India, where the maximum incidence usually falls in October; plague was much more prevalent in the Bombay presidency than at the same time last year; from August 23 to September 19, there were 3,543 deaths from plague in the provinces of India. An increase in plague is also reported in Java and Siam, but in the principal ports of Asia, the plague situation was good during October; there were sporadic cases in September and October in Greece and in Egypt; there was an outbreak in June in Nigeria, which up to the middle of October amounted to 407 cases, with 301 deaths.——The cholera epidemic in Shanghai reported recently seems to have ended, only one new case being reported during the last two weeks in October. The outbreak in Manila which decreased in October had amounted to 199 cases and sixty-nine deaths at the time of this report. The severe epidemic in Kashmir had caused 11,400 deaths up to September 19, being exceeded only by the outbreak in September, 1919. The Punjab was infected from Kashmir (which is rare) and there were 2,600 deaths from cholera there up to September 19.——Yellow fever has been more prevalent on the Guinea coast than in 1924.——Typhus continued to decline in eastern and central Europe during the autumn, the incidence being the lowest since the World War; no cases were reported this year in Austria and only two in Germany; there were fifty-one cases and two deaths in Poland in the month ending October 3.——Malignant smallpox has been extremely rare in Europe except in Spain, no death having been reported since June in any other European country. Smallpox caused 3,572 deaths in Mexico in the first eight months of 1925, the greatest prevalence being in Central Mexico. Smallpox overran most of India during the first half of this year; an outbreak seems to occur here every fifth year.——There was an increase in mild influenza in October in England and Wales, with 196 deaths in the four weeks ending October 17 as against sixty in the preceding four weeks. British Guiana had an outbreak of 650 cases of influenza in the month ending August 8; the disease seems to be affected little by seasonal influences, an outbreak occurring about every nine months.——Scarlet fever is on the increase in central Europe and Great Britain; there is no indication, however, that the epidemic will be more extensive than those which occur every few years, the last one being in 1921.——The general mortality has been favorable in European and North American cities, and is the lowest on record in German cities, and markedly improved in other parts of central Europe. Mortality, which in the last century was often excessive during the summer, is now usually the lowest at that time except in subtropical countries. Modern sanitation has been less successful in dealing with winter excess mortality, which is caused largely by influenza and other respiratory diseases.

FOREIGN

Personal.—Dr. William E. Gye has been awarded the annual fellowship established by London University for the graduate who has done most for the advancement of medicine in the last five years.

Campaigns Against Quacks in Japan.—The *Japan Medical World* states that the metropolitan police of Tokyo are now enumerating all quack doctors in that community, preparatory to launching a campaign to enforce the law against them. While investigations in October revealed only eighty-eight quacks practicing without a proper license, current reports place the number at several hundred. The law exacts only a fine of 500 yen for practicing without a license, which the quacks are said to be able to pay without much inconvenience.

Professor of Physiology at Cambridge Appointed.—Dr. Joseph Barcroft was appointed professor of physiology of Cambridge University in England, December 16. Professor Barcroft was president of the physiologic section of the British Association in 1920; was awarded the Royal Medal in 1922 and the Baly medal in 1923; he was the Oliver-Sharpey lecturer, Royal College of Physicians, in 1915; the Lowell lecturer in 1922; lecturer in Kings College, Cambridge, and reader in physiology in Cambridge; Fullerian professor of physiology at the Royal institution, and editor of the sixth edition of Huxley's Physiology.

Infant Mortality in Bombay.—In his last annual report, the health officer of Bombay showed that the infant mortality in that city in 1924 was 419 per thousand births, although, if the rate is calculated on the deaths of infants born in Bombay, it is reduced to 275, as 3,159 of the deaths were of infants born elsewhere. "This excessive emigration of the infants is due," says the *Medical Officer*, "to a custom among native women of going to the country for confinement. The chief causes of death were diseases of the respiratory system, debility and premature births, convulsions, diarrhea, smallpox and febrile diseases; other adverse factors were poverty, ignorance, malaria, and the practice of soothing babies by the administration of opium."

Death of Sir Richard Powell.—Sir Richard Douglas Powell, one of the king's physicians, died in London, December 15, aged 83. Dr. Powell became physician in ordinary to Queen Victoria in 1889, to King Edward in 1907, and to King George in 1910. He was emeritus lecturer on medicine in Middlesex Hospital, consulting physician to Brompton and Ventnor hospitals, and former president of the Royal College of Physicians; he delivered the Harveian oration in 1914 on "Advances in Knowledge Regarding the Circulation and Attributes of the Blood Since Harvey's Time"; was the author of "Diseases of the Lungs and Heart" and other publications, and had been a Knight of Grace of the Order of St. John of Jerusalem since 1898.

Government Services

Annual Report of Public Health Service

In the annual report of the U. S. Public Health Service submitted to Congress, Surgeon General Cumming states that preliminary figures indicate a death rate for the country in 1924 of about 11.9 per 1,000, as compared to 12.4 in 1923; with 17.6 in 1900 and with 19.8 in 1880. Heart disease ranks first in the United States as the cause of death and its incidence is steadily increasing. Influenza and pneumonia combined occupy third place. Infant mortality has shown an appreciable decrease, but the number of deaths of mothers incident to childbirth has shown little change in the last nine years for which data are available. From 1915 to 1922 inclusive, it is estimated that for every 100,000 babies born, the lives of from 600 to 900 mothers were sacrificed; about one-third of these deaths were caused by infection of the mother at childbirth. The diphtheria death rate shows a fall from 43.3 per 100,000 in 1900 to 12.1 in 1923. "If parents could be induced to protect their children by the use of the 'toxin-antitoxin' process of immunization," says Surgeon General Cumming, "there is no reason why diphtheria might not be still further greatly reduced or even exterminated." The death rate from tuberculosis in the registration area dropped from 201.9 per 100,000 in 1900 to 93.6 in 1923. Reports from thirty-five states having a population of nearly eighty-eight and one-half millions give a death rate of 88.6 for the calendar year 1924.

Reports from thirty-five states for the calendar year 1924 showed an increase of 75 per cent in the number of cases of smallpox and 628 per cent in the number of deaths as compared with the year 1923. The smallpox case-rate in the United States is at present, according to reports received, the highest of any civilized country in the world. It may be said, however, that much of the disease is of a mild type and in some countries many of these cases would be classed as "alastrim." It is also believed that smallpox is better reported in the United States than in most other countries. Eleven cases of tetanus, most of them fatal, occurred in the United States during the year as a result of the use of bunion pads as vaccination dressings. Efforts are being made to induce physicians to follow a standard technic when vaccinating, and warnings were issued against the use of shields and bunion pads as dressings.

Foreign Letters

LONDON

(From Our Regular Correspondent)

Dec. 5, 1925.

Preservatives in Food

Mr. Neville Chamberlain, minister of health, speaking at a luncheon of the provision trade section of the London Chamber of Commerce, said that he recognized that the new regulations regarding the use of preservatives in food (THE JOURNAL, September 26, p. 988) might involve inconveniences, even some hardship, to individual members of the community, but the very last thing the government desired was to interfere with trade or industry. The crux of the controversy that had arisen was the plea for the removal of the prohibition of boron compounds. He would explain the considerations which had weighed with him in this matter. It was known that these boron compounds were poisonous, that they readily passed into the blood stream and were not excreted so quickly as they were absorbed, and they therefore had a cumulative effect. During the past forty years their use had been increasing, and there had been an increase in the prevalence of certain digestive diseases, such as appendicitis and dyspepsia. The retort might be that it could not be proved that these evils were due to boric acid; he could not prove it, nor could the traders prove that they were not. Before the cancellation of the prohibition could be urged, it was necessary to prove that this form of preservation was necessary, for if it was not necessary there was no point in the demand for further inquiry as to the effects of boron compounds. The trade had also to take into account that in certain countries the use of these preservatives was absolutely forbidden, except in a certain limited number of foods; and in all cases, although their use might be general, there were certain firms that had succeeded in doing without them. The case for prohibition was therefore unanswerable. There was considerable time to make the necessary adjustments and to consult with the transport companies and shippers.

Arsenic in American Apples

Shopkeepers in a London district were summoned for selling apples found to contain arsenic, in one case to the amount of one-fifth grain (0.13 gm.) to the pound (453 gm.). Owing to a case of illness reported to the local health authority as due to eating apples, samples were taken. Five of the samples of Jonathan apples imported from America were found to contain arsenic; in each case, more than a hundreth of a grain to the pound. Samples of English apples, taken in the district, were free from arsenic. It was pointed out that fruit growers in all parts of the world made a practice of spraying trees for the purpose of combating insect pests and that in America not only the blossom but also the fruit was sprayed on the tree. For the defense it was stated that in America the presence of the dreaded fruit tree pest known as the codling moth made it necessary to spray fruit trees with arsenic. The season had been unduly dry, with the result that the arsenic had not been washed off. Although the shopkeepers could not be expected to know anything about the arsenic they were, according to the law, fined in each case to the extent of $13, with $5 costs.

The Prevention of Venereal Diseases

Sir Arthur Newsholme, who for many years was principal medical officer to the ministry of health, delivered a lecture for the British Social Hygiene Council on "The Community and Social Hygiene." He was convinced that the control of venereal diseases should become a question of organizing public opinion and action against irregular sex relations.

For practical purposes these were solely responsible for such diseases. He regarded the general advocacy of prophylaxis in irregular sex relations as inimical to moral progress, by means of which, in conjunction with extended medical measures, we should render venereal diseases a shadow of their present self. In recent years, and particularly in the last decade, notwithstanding the terrible sexual lapses due to the Great War, the hope of controlling venereal diseases had become increasingly bright. Much good could result from the greater frankness with which sexual problems were discussed. He regarded public propaganda on preparations for prophylaxis in irregular sex relations as a sort of partnership in the projected immorality and as an encouragement of promiscuity. In many departments of life there had been vast improvements in our sense of social responsibility. In sexual morality there was indubitable proof of improvement. One of the problems was to ascertain how an act, which even now was regarded as venial in a man and infamous in a woman, could by force of public opinion be made equally infamous for the two sexes. Facilities should be given to teachers to attend special courses of lectures on social hygiene and ethics, and conferences should be held between teachers and parents. Biology, psychology and hygiene should be taught in all secondary schools. Sexual hygiene should take its place with other branches of public health education directed toward self-control as affecting health.

Illicit Traffic in Narcotics

The extent of illicit traffic in narcotics, such as opium, morphin and cocain, is revealed in a report issued by the advisory committee on traffic in opium of the League of Nations. More than 1,100 seizures of contrabrand drugs have been reported to the league during the last four years. In the majority of cases seizures were made on board ships, where drugs are concealed in the most unsuspected and out of the way places, as, for instance, the lamps of the bridge compass, the water tank under the boilers, in barrels of smoked fish, casks of phenol (carbolic acid), tea chests with false bottoms, the cylinders of a life raft, under coils of rope in the wheel room, or in the soles of the shoes of the accused. In other cases the drug has been shipped as phonograph records or electric bulbs, or described as "salted pickles" or cocoa powder. Two large consignments of drugs were concealed in iron safes and iron beadsteads, respectively. The methods of concealment are most ingenious and involve considerable expense. Heroin in bedsteads seized at Hongkong was packed in tin tubes, specially made to fit the hollow legs of the bedsteads. Below the tubes, plugs had been soldered into the hollow legs, and below these the casters were screwed on.

MADRID

(From Our Regular Correspondent)

Nov. 12, 1925.

Medical Reciprocity with Foreign Countries

The army directory has issued a decree providing for reciprocity with foreign countries as regards the practice of the several professions, including medicine. Heretofore, it has seemed strange that this country should grant all kinds of facilities to foreigners, while Spanish physicians find so many handicaps when trying to practice abroad. The situation was the worst in precisely those nations in which large numbers of Spaniards make their homes. In fact, medical practice was practically prohibited to Spaniards, except in the rare cases in which physicians had studied there from grammar school on. Thus, in neighboring France, Spanish physicians can be counted on the fingers of both hands, and those few are the sons of emigrants or persons who have spent their entire lives there. Nationalist sentiment has been so aroused that a number of Spaniards, urged to settle in France during the World War, were compelled to leave the country afterward; forbidden to practice, in peace times, the very profession which they had been called over to aid in.

The present reciprocity measure had been demanded on several occasions by medical societies in the provinces bordering on France, as they have been the ones suffering most from the influx of foreign physicians. The government has now complied with their wishes, but has recognized the rights of those physicians settled in Spain before official publication of the new provisions.

A proof of the indifference shown by physicians even to their most vital problems is found in the scarcity of comment on such a far-reaching question. Only two articles have discussed the matter. In one, Dr. Cortezo, the editor of *El Siglo Médico* and president of the council of state, pointed out the possible danger of a rush of foreign physicians. Germany and Austria may now accept reciprocity, as their universities keep on training physicians while the loss of territory has decreased the available fields. Thus the German Reich could send 100 graduates to Spain for each Spanish physician going to practice in Germany.

In the other paper on the same subject, Dr. A. Fernández de Velasco regrets the lack of reciprocity agreements with Latin American republics. This point seems to have been overlooked in the recent decree. Among Latin American countries, only Bolivia, Colombia, Costa Rica, Honduras, Nicaragua, Peru and Salvador have so far signed such treaties. Panama is now considering the matter. The most important republics, such as Argentina, Chile, Cuba and Mexico, not to mention Ecuador, Guatemala, Paraguay, Santo Domingo, Uruguay and Venezuela, have taken no action on this matter. Reciprocity treaties prove influential in promoting university relations, as shown by the fact that the two countries having most students in Spain are Colombia and Peru, both of which enjoy reciprocity rights, while hardly any students come from the others. In Paris, there are nearly 7,000 Latin American students, and 20,000 Latin American families have trailed them there, spending millions of francs each year. In Spain, there are barely 200 Latin American students. Argentina shirks reciprocity on the ground that it has already an overflow of graduates. In a single Argentine medical school, that at Buenos Aires, the enrolment reaches 5,000. Cuba also declines reciprocity, alleging that if this privilege is extended to Spain, France and Italy will expect a similar courtesy. Porto Rico and the Philippines are out of the question, because of American control.

Pediatric Congress

The Spanish Congress of Pediatrics met with great success. A welcome departure was the active participation, in a medical meeting, of noted representatives of other professions, such as lawyers, judges, teachers and priests.

One of the most interesting papers was by Dr. González Alvarez on child welfare. He protested against the undue emphasis laid on the care of illegitimate children rather than of children in general. González Alvarez pleaded for compulsory breast feeding, a conclusion accepted by the congress only so far as it applied to mothers in lying-in hospitals. His other resolution, forbidding outside nursing by mothers who did not nurse their offspring, received endorsement. González Alvarez drew a somber picture of present Madrid employment agencies for nurses. These agencies are usually boarding houses. The rooms are small and overcrowded. Feeding utensils are promiscuously used. In order to keep the milk flow of unemployed wetnurses, two or three overgrown indigent infants are used. These children usually have rickets or scrofula, if not syphilis. If there are no children on hand, the other women replace them. In one recent case, the nursed baby acquired itch from one of these nurses. In another

nurse, skin and mucous signs of syphilis were detected during her first month's employment. After dismissal, such nurses return to the employment agency to continue spreading disease, as no medical examination is enforced, and the provision requiring nurses to have health cards has long been neglected. In order to remedy such deficiencies, González Alvarez suggested the passage of a law, but with "teeth" in it, so that child health should really be protected.

Dr. Bandelac de Pariente of Paris stressed the advantages offered to young and visiting physicians in France by Umfia, the Franco-Iberic-Latin-American society.

The one mishap attached to the congress was the sickness, contracted during the meeting, of the surgeon Arquellada, who died a few days after his return to Madrid. Although still young, Dr. Arquellada, surgeon at the Niño Jesús (Children's) Hospital of Madrid, had made a name for himself through the introduction of several surgical methods. These he described in the child surgery congress held recently in Paris.

Farewell to a Clinical Professor

The Madrid Medical School witnessed recently an almost unprecedented scene, when Dr. A. Redondo, the professor of pathology and clinical medicine, reached the retirement age. His last lecture was attended by former pupils from all over the country, some of them now occupying high positions. Dr. Redondo was satisfied to be just a professor. He never tried to go into politics or to devote his talents to social problems. His only acquaintances were among his pupils, who now have shown the love and respect they feel toward their old preceptor. When the lecture was over, the whole audience surrounded the professor and went with him as far as the door of the school.

Fifty Years of Professional Life

For fifty years, Dr. S. Cardenal of Barcelona has practiced medicine and surgery. The authorities and scientific societies of his home town thought the occasion had come to show their recognition of such a useful career, which included the introduction of antisepsis in Spain. For the first time in the history of the city, such honors have been rendered to a physician. Dr. Cardenal's name was given to the hospital clinic where most of his surgical work has been done, and a number of meetings were held, with no end of laudatory speeches. Fortunately, Dr. Cardenal was strong enough to bear up with so much enthusiasm. As a practical way of showing his gratitude, he has endowed a fellowship, which will permit some meritorious young man to become a physician.

Aid to Physicians

Dr. Murillo, director general of public health, has issued an order that will secure indigent physicians free admission to the private ward at any government hospital. Heretofore, impecunious physicians in need of medical treatment had often been unable to secure admission to hospitals, as all free beds available were occupied.

First Spanish Medical Salon

A Madrid paper, *Vida Médica*, had the original idea of holding an exhibition of art works by physicians. A prerequisite was that the subject should not be medical. There were more than 300 entries, including paintings, sculptures, architectonic plans and even literary works and musical compositions. The exhibition was held in the rooms of the Society of Friends of Art, one of the most elegant and aristocratic places in town.

The king personally inaugurated the show, being accompanied by the whole army directory and the diplomatic corps. He proved himself a good critic in passing judgment on the exhibition. "I thought this exhibition would be rather humorous, but I see here a number of interesting works." The satirical writer A. Azpeitia has given expression to the alleged displeasure of patients at seeing physicians thus go into strange fields. "Physicians neglect their profession and devote themselves to dilettantism while we suffer. Physicians may go in for all kinds of amusements, provided they keep it quiet. Patients are naturally selfish and want to monopolize their physicians' attention. They do not want even to share it with other fellow sufferers."

On the other hand, Dr. Alvarez Sierra said: "There is no profession or class having so many receptive elements for esthetic emotion as physicians. . . . Every physician carries within him an artist's soul. Such artistic manifestations are safety valves offering an outlet to the nervous tension of those constantly walking in the footsteps of death." Dr. Juarros endorsed this view: "Pain, among its many qualities, has the power to take off all social masks. That spectacle of human zoological realism would embitter life, were it not for a romantic reaction on the part of the physician."

There is little doubt that this first attempt proved quite meritorious and compared favorably with other art exhibitions both here and abroad. If physicians now expressing their originality in lay fields ever devote a similar effort to their own profession, their success will prove astounding.

BELGIUM

(From Our Regular Correspondent)

Nov. 18, 1925.

Cerebrospinal Meningitis

From his experience in the army of occupation, Dr. Muschang points out some facts concerning symptomatology of cerebrospinal meningitis. In addition to the outstanding symptoms, there are irregularity of the pulse, herpes labialis and petechial purpuric eruptions. In infants, abnormal tension of the fontanels is of high diagnostic value. Ocular signs, rapid coagulation of the cerebrospinal fluid and xanthochromia are of great importance. From the therapeutic standpoint, he recommends: (1) injection of serum into the vertebral canal; (2) prompt action; (3) large doses; withdrawal of 50 c.c. of cerebrospinal fluid, injection of 40 c.c. of serum, this dose to be repeated systematically for four days and combined with 40 c.c. of serum intramuscularly, and (4) specificity in the serotherapy: polyvalent serum, and, as soon as the laboratory has identified the causative meningococcus, injection of anti-A, anti-B or anti-C serum.

Free Tours to the Congo for Young Physicians

The *Bruxelles médical*, aided by a transportation company, has for some time been providing an opportunity for a number of young physicians and senior medical students to visit the Congo region. The young men selected are given a free passage in return for assisting the ship's physician.

Tartar Gingivitis in Relation to Cancer

Before the Société belge d'otorhinolaryngologie, Dr. Heyninx recently spoke on the concomitance of cancer of the respiratory and digestive tracts with alveolodental pyorrhea and gingivitis. There may be a relation of cause and effect between the two affections. Also, cancer of the tonsil may be secondary to a primary gum affection or the like. If the gum affection were of microbic origin, it might produce chronic irritation and result in hyperplasia of the mucosa which covers the tonsil. If neoplastic in origin, it would produce metastasis in the tonsil. Infected saliva might carry the virus into the throat and esophagus.

Resuscitation of the Heart

Dr. Schepens, in addressing the Société de médecine of Ghent, submitted a study of forty-three cases of reanimation

of the heart by injection of epinephrin, twenty-three of which were successful. He concludes that this should become a routine method and that the surgeon should always have the necessary material at hand. Dr. Schepens gives, as conditions of success, not to lose precious time; to begin with a small dose (from 0.25 to 0.50 mg.), and not to interrupt artificial respiration.

Surgery of Ureteral Calculus

At the annual session of the Société d'urologie, several authors reported cases in which ureteral calculi had been removed by the extraperitoneal route. François and Tant reported several cases in which low position of the calculus makes possible the paraperitoneal route. The location of the calculus is determined by means of roentgenography and the opaque ureteral sound. Van den Branden referred to the vaginal route for the extraction of calculi.

Spina Bifida and Incontinence of Urine

Addressing the Société belge d'urologie, Dr. François pointed out the significance of spina bifida as a cause of incontinence of urine. Patients with incontinence of urine should be subjected to a roentgenologic examination with a view to detecting spina bifida of the fifth lumbar and first sacral vertebrae and metamerization of the sacral vertebrae. The discovery of malformation should be followed by epidural injection of iodipin through the sacrococcygeal hiatus or between the arches of the first lumbar vertebrae. It should then be determined whether or not the iodipin comes to rest near the site of the malformation.

In case of spina bifida and metamerization, with arrest of the iodipin at the level of the malformation, if the iodipin does not effect a great improvement, laminectomy of the sacral vertebrae and of the fifth lumbar vertebra, together with removal of abnormal processes (yellow ligament, fibrous adhesions, lipoma) that compress the dural culdesac may be considered if the patient is an adult. If the patient is a child, however, surgery should not be undertaken lightly—in any event, all other forms of treatment should be tried first.

The Problem of Heredity

In connection with the reopening ceremonies of the University of Brussels, Dr. Brachet, rector of the university, delivered an address on the present and future aspects of the problem of heredity. Numerous species of animals have become extinct. Evidences of previously unknown species are being discovered every day. Scientists have been able to classify animals and plants in accordance with a probable order of filiation. Some hold that reconstruction of the history of animals is possible. To be sure, methods now available for the construction of an historical biology are open to suspicion or nearly exhausted. It is the present living being that must be the object of research.

A Cruise in the Levant

The *Bruxelles médical* organized, some time ago, a successful Mediterranean tour. The Brussels journal is planning a second Mediterranean tour for the early spring. This voyage will take on an international character. The cost of the trip for physicians will be about 4,000 francs ($160).

Dr. Louis Coty and the Museum of Public Health

In the cemetery at Jemappes, a monument has recently been erected over the grave of Dr. Louis Coty, who was an active pioneer in public health work. Nearly twenty years ago Coty established the dispensary for ancylostomiasis, which has aided in eradicating that terrible disease, and the Museum of Hygiene at Mons, an organization for the education of the people. Museums usually contain monotonous exhibits which fail to attract the passer-by. The museum of Mons, however, essays to give practical instruction in problems affecting dwellings, foods and education. Numerous samples and some very significant pictures indicate the respective nutritive values of the essential food products.

The Society of Legal Medicine

At the reopening session of the Société de médecine légale of Belgium, the newly elected president, Professor Heger-Gilbert, took up the problem of stabilization of functions of medicolegal experts in courts, together with revision of fee schedules applicable to such experts. Suggestions for the modification of the present schedule were addressed to the Commission des frais de justice répressive.

BUCHAREST

(From Our Regular Correspondent)

Nov. 18, 1925.

Infant Feeding

In speaking before the Medical Society of Oradea Mare on infant feeding by means other than its mother's milk, Dr. Bálint distinguished between what he calls "substitute" and "artificial" feeding. The former he regards as feeding with milk from another woman or after modification from some other animal; the latter he defined as feeding with preparations artificially manufactured from milk or from other products. He gave an account of the laboratory methods of modifying milk in accordance with physicians' prescriptions. Artificial feeding is condemned by Bálint, and the majority of foods on the market are strongly decried. The great evil of them all is the ease with which they can be used. They are readily digested, but this is largely because they contain so small an amount of food material. Disorders arising from imperfect feeding escape for a long time the notice of the parent. Such children are pallid, flabby and listless, and lack resistance against disease. The market is flooded with infants' foods, all more or less likely to produce rickets and other nutritional diseases, and their banishment from nurseries would greatly aid the development of the race.

Hygienic Dwellings for Working Men

Hygienic dwellings for working men and chiefly for agricultural laborers in Roumania have been provided since 1919 by means of government grants, a sum having been included for that purpose in each year's budget. The annual expenditure from 1919 to 1924 amounted to $15,000, but the demand for cottages grew until this year the annual grant was increased to $24,000. As it is certain that this amount also will prove inadequate, an act will be passed that will authorize the conversion of $24,000 capital into interest, thus making it possible to build about 1,000 cottages yearly. Another clause of the same act provides that in the case of land being sold by the local authorities to laborers for building purposes, all the necessary plans, surveys and contracts are to be made by the minister of agriculture at state expense. The interests of the laboring classes are safeguarded by the act in a variety of other ways. For instance, land selected for building purposes must be suitable as regards soil, air and drinking water; and speculation by the builder or contractor is forbidden. Conditions of the sale or loan, terms of the loan and the general plan must be favorable to the laborer. During the period allowed for repayment, the property cannot be seized for debt, divided, encumbered or alienated in any way; exemption from taxation is granted for fifteen years. Preference is given in selection of sites to applications coming from localities which make a free grant of land either for house or for gardens. Materials are forwarded at cost price on government railways, but state assistance is granted only for buildings designed to accommodate one family. Each cottage occupies a plot of from 1,000 to 1,200 square

yards, and the cost of building varies from $200 to $350. Loans may be repaid in annual instalments, extending over twenty or twenty-five years. Under the act of 1920, 7,500 workmen's cottages were built in one year. Another result of this act has been to abolish land speculation, and to keep down the cost of building sites. Owing to the fact that most Roumanian parishes own the adjacent country, which formerly was used as common land to supply fuel and pasturage, this land is now convenient for the erection of workmen's dwellings.

But the care of the state is not confined only to the housing of agricultural laborers. A law has been passed which authorizes the construction of houses in the larger cities for employees of both state and private enterprises, the government undertaking the building of the houses, setting aside a sum for that purpose. Actual construction will begin in 1926. The average building cost of a two-room flat is $640; the average yearly rental amounts to $50. A flat of three rooms and kitchen costs from $950 to $1,000, and may be rented for $60 a year.

Psychoses Associated with Migraine

At the Medical Society of Cluj, Dr. Jacobi read a paper on psychoses associated with migraine. He said that simple hemicrania sometimes develops mental irritation, depression, anger or slight hallucinations. With migraine of an epileptic type a visual aura can precede an attack. The true epileptic nature of migraine is noted when the visual aura becomes red. Abortive attacks, psychic symptoms or a psychic equivalent can occur. A transitory psychosis sometimes follows or accompanies migraine. This is important as indicating the necessity of antiepileptic treatment for migraine. Dr. Jacobi notes close clinical relationship between migraine and hysteria major. Several types of cases are on record; among them: (1) a combination of hallucinations of sight, confusion, amnesia or ataxia or aphasia, with attacks of migraine lasting six hours; (2) confusion, with religious delirium; (3) hemicrania, with temporary blindness, paresthesia and a speech disturbance, and (4) hemicrania after mental shock, with anxious delirium and disturbance of consciousness for three days. The occurrence of these psychoses is rare.

Detection of Yeast Extract as an Adulterant of Meat Extract

The *Revista Farmaciei*, a pharmaceutic journal, publishes a method by which one is enabled to detect as little as 1 per cent. adulteration of meat extract with yeast extract. This may act as an efficient check to dealers working off stocks of yeast extract as genuine meat extract. Such stocks are known to have been hurriedly withdrawn from the market since the test was published. If the sample gives a doubtful or negative result by that test, but is still open to suspicion, one may take from 3 to 6 gm. and dissolve it in 3 or 6 c.c. of water, according to the quantity taken; to this one may add sufficient spirit—methylated will answer the purpose—to throw down all that is insoluble in alcohol. After a vigorous shaking, one may separate the insoluble residue by decanting or filtering, dissolve this residue in 60 c.c. of water, filtered if necessary, and proceed as before. If the yeast extract is present, the characteristic bluish-white precipitate will be thrown down, on boiling with the modified Fehling's solution; this may be collected and weighed.

Government Attack on "Patent Medicines"

The postoffice department of Roumania has decided to establish a censorship over newspapers in an endeavor to prevent publication of offensive or fraudulent advertising. Newspapers printing such advertisements will be barred from the post. The advertisements aimed at are chiefly those of the lowest class of "patent medicines," about fifteen of which are at present being examined by the chemical laboratory of the ministry of sanitation to see whether they correspond with the claims made. This censorship has been in existence in Austria for some time, and has long been advocated in the medical journals as a public necessity.

VIENNA

(From Our Regular Correspondent)

Nov. 17, 1925.

Observations on Brain Abscesses

Dr. Winckelbauer recently addressed the Vienna Gesellschaft der Aerzte on the brain abscesses observed by Dr. Brunner and himself at the Eiselsberg clinic. He emphasized the frequency of recurrences and the consequent gravity of the prognosis. Frontal brain abscesses are adjacent to the accessory sinuses of the nose. Injuries of the frontal region are accordingly dangerous, as was observed in two cases that ended fatally in spite of repeated operations. In both cases pus had been retained but was not discovered at the repeated operations. Such abscesses should be drained at the lowest point, so that pus retention may be prevented as far as possible. An abscess sometimes takes as much as five weeks to develop, while in other cases it is manifest on the third or fourth day. In the discussion that followed the address, Dr. Jerusalem stressed the need of early diagnosis and early operation, as was shown by the experiences of the war. Otogenous abscesses, particularly those of the frontal and temporal lobes, as discussed by Alexander, are more favorable than abscesses of the cerebellum. The mortality from operations in frontal abscesses and those of the temporal lobe ranges from 40 to 50 per cent., while in cerebellar abscesses it runs as high as 90 per cent. Abscesses of the temporal lobe are drained from the middle ear, the lowest point; but cerebellar abscesses can be operated on only from the side. Otologists also extol drainage from the lowest point. Beck held that the variety of pyogenic agent is important in the prognosis. It is his experience that fetid, green, crummy pus offers a more grave prognosis than pus mixed with mucus. In the latter case a pyogenic membrane is present, so that the abscess can be better controlled.

The Seventieth Birthday of Professor Dimmer

The medical department of the University of Vienna celebrated, a few days ago, the seventieth birthday of Prof. Dr. Dimmer, an authority in ophthalmology. He studied under Nolte and Jäger, and served as an assistant under Fuchs. In 1885, he was called to a chair in Innsbruck; in 1900 he removed to Graz, and in 1910 he became Schnabel's successor in Vienna, where he took over the management of the First Eye Clinic. In 1918 Professor Fuchs retired and Dimmer succeeded him, while Meller assumed Dimmer's previous position. Dimmer's principal activity in ophthalmology has had to do with the normal and the diseased fundus, and, more particularly, with the histologic interpretation of many conditions not explainable solely with the ophthalmoscope. His researches have been on the detailed structure of the macula, its reflexes and the reflexes of the fundus as a whole. In the controversy with Gullstrand over the color of the macula, he appears to have been victorious. Photography of the fundus was first developed by Dimmer; his apparatus gives good service. His most important publication is "Der Augenspiegel," which is a complete treatise on modern ophthalmology. As a teacher, he stresses exact correction of the eyes, and his lectures are a model of exactness; likewise his operations betray almost pedantic exactness. As Professor Dimmer himself—possibly by reason of excessive modesty—resisted all urgings to participate in the festivities, his natal day was celebrated in his absence, by his many friends, admirers and colleagues.

Accidents Due to Electricity and Treatment in Electric Shock Simulating Death

In a lecture forming a part of the Vienna "Seminar-Abende" for general practitioners, Professor Jellinek discussed recently the etiology, pathology and treatment of accidents due to electricity. He attaches great importance to insulation of electrical household utensils, as, through short circuits, accidents may easily arise even with a low voltage (from 110 to 220 volts). Lamps, flatirons, cooking utensils, and, more recently, radio apparatus have been the cause of such accidents. Injuries actually due to electricity are not true burns; only an arc light can produce burns. Wounds due to electricity are generally grayish white, circular and firm, without a trace of reactive inflammation in the adjoining area. The hairs next the wound are not even singed, and, if the patient survives, the wounds are not in the least painful. There is not always a continuous lesion and almost never a severe or suppurating infiltration of the surrounding area—not even in injuries of the bones and joints. After a latent stage possibly of several weeks, an aseptic necrosis sets in, without any general manifestations, even though there has been extensive loss of substance or so-called spontaneous amputation. If such injuries are treated conservatively, the prognosis is very good. Surgery is contraindicated for two reasons: (1) during the first few days or even weeks, it is impossible to determine the extent to which degeneration of the tissues may occur; (2) the vessel wells, even at some distance from the electrical injury, sometimes are very vulnerable, so that severe hemorrhages may be induced. Scar tissue after such accidents is soft, is vascular and never shrinks, and is thus very favorably constituted. The strength and character of the current are not the sole factors in determining the intensity of the injury. The range of individual susceptibility is great; it varies in the same individuals on different occasions. What is termed "death from electricity" is, as the Vienna school has shown, in reality only apparent death. Necropsy reveals only agonal phenomena, such as edema of the brain or lungs and extravasations, but no explanation for the fatal outcome is found. Jellinek thinks that disturbance of function of the vital organs causes death. Support for this belief is found in the fact that persons who survive electrical accidents never show evidence of loss of organs even when they have lain like dead for hours. If the patient recovers, his restoration is rapid, once it begins. The sovereign remedy is artificial respiration, but this remedy must be applied as soon as possible after the accident and may need to be continued for many hours. Until death spots appear, there is still hope, even though the current was very heavy. All this is true likewise of injuries from lightning. Cases are recorded in which the first attempts at resuscitation were begun a full hour after the patient was struck by lightning, while another hour passed before the efforts were successful.

Trachoma, Anthrax and Typhoid in Austria

During the last few weeks, there have been three epidemic outbreaks of infectious diseases in the immediate vicinity of Vienna. The recent outbreak of typhoid in Neunkirchen has involved thus far ninety-two cases and has resulted in five deaths. The source of the infection was the milk from a small dairy. Two carriers came in contact with the milk. The outbreak of trachoma occurred in a factory near Vienna, in which not only native workmen but others from Slavic territory were employed. About twenty patients were hospitalized, and the factory was closed; this ended the outbreak. The epidemic of anthrax, to be sure, comprised only seven cases, but the infection had been carried into several districts. The infection started in a brush factory, and the unusual character of the disease gave rise to a certain degree of laxity, but the authorities soon got things in hand.

Marriages

WALTER PETERS GARDINER to Miss Catharine Marion Sullivan, both of New Orleans, December 2.

RUSSELL ETHELBERT PALMER to Miss Suzanne Brisbois, both of St. James, Mich., in November.

GEORGE ELISHA MAY, Boston, to Miss Winifred Arnold of Wyoming, N. Y., September 24.

STEPHEN JAY SPITZER, New York, to Miss Elizabeth Noyes of Oakland, Calif., December 9.

Deaths

Henry Redwood Wharton ✙ Berwyn, Pa.; University of Pennsylvania School of Medicine, Philadelphia, 1876; member of the American Surgical Association; formerly clinical professor of surgery at the Woman's Medical College of Pennsylvania, Philadelphia; at one time on the staffs of the Presbyterian, Children's and Bryn Mawr (Pa.) hospitals; author of "Textbook on Minor Surgery and Bandaging"; aged 72; died, December 3.

George French Little ✙ Brooklyn; Medical Department of Columbia College, New York, 1895; member of the American College of Physicians; formerly assistant clinical professor of pediatrics, Long Island College Hospital; at one time on the staff of the Kings County Hospital; aged 60; died, December 5, at the Methodist Episcopal Hospital.

William Eck Doran ✙ Colon, Mich.; University of Michigan Homeopathic Medical School, Ann Arbor, 1904; formerly president of the board of education; served in the M. C., U. S. Army, during the World War; aged 46; died, December 6, at the Sturgis (Mich.) Memorial Hospital, of erysipelas.

Henry Tierney Bray, New Britain, Conn.; University of Vermont College of Medicine, Burlington, 1902; member of the Connecticut State Medical Society; chairman of the board of health; on the staff of the New Britain Hospital; aged 46; died, December 7, following an appendectomy.

Lewis Cass Taylor ✙ Springfield, Ill.; Bellevue Hospital Medical College, New York, 1875; past president of the Illinois State Medical Society; secretary, state department of registration and education; formerly member of the state board of health; aged 71; died, December 14.

Walter Gohring Ryon ✙ Poughkeepsie, N. Y.; Columbia University College of Physicians and Surgeons, New York, 1896; member of the American Psychiatric Association; superintendent of the Hudson River State Hospital since 1917; aged 51; died, December 4.

Albert E. Acker, Jacksonville, Fla.; Georgetown University School of Medicine, Washington, D. C., 1907; member of the Florida Medical Association; served in the M. C., U. S. Army, during the World War; aged 61; died, November 25, following a long illness.

Lavine Alonson Bromley, Granville, N. Y.; University of Vermont College of Medicine, Burlington, 1882; member of the Medical Society of the State of New York; aged 68; died, December 2, at the Glens Falls (N. Y.) Hospital, of heart disease.

Amos H. Horn, Putnamville, Ind.; University of Virginia Department of Medicine, Charlottesville, 1883; member of the Indiana State Medical Association; aged 68; died, December 6, at the Putnam County Hospital, Newcastle, of pneumonia.

John Rogers Parry, Jr., Philadelphia; University of Pennsylvania School of Medicine, Philadelphia, 1911; served in the M. C., U. S. Army, during the World War; aged 35; died recently, at the Misericordia Hospital, of pericarditis.

A. A. Smith, Hawkinsville, Ga.; Savannah Medical College, 1875; past president of the Medical Association of Georgia; for eight years member of the state board of medical examiners; Civil War veteran; aged 78; died, November 26.

Howard Eugene White, Logan, Ohio; College of Physicians and Surgeons, Chicago, 1888; for eight years postmaster of Fairmont, Ill.; formerly bank president; aged 61; died, December 1, of pernicious anemia.

Henry M. Moulton ✙ Cumberland Center, Maine; Dartmouth Medical School, Hanover, 1889; Medical School of Maine, Portland, 1889; formerly member of the state legislature; aged 61; died, November 22.

Charles E. Rogers, Gray Court, S. C.; University of Tennessee College of Medicine, Memphis, 1886; member of the South Carolina Medical Association; aged 67; died, December 3, of cerebral hemorrhage.

Charles Harrison Lee, Fayette, Mo.; Barnes Medical College, St. Louis, 1893; member of the Missouri State Medical Association; aged 52; died, November 28, at St. Joseph's Hospital, Kansas City.

Michael Joseph Burke, New York; Medical Department of the University of the City of New York, 1875; member of the Medical Society of the State of New York; aged 75; died, November 27.

Percy Eugene Riley ✠ Eau Claire, Wis.; University of Minnesota College of Homeopathic Medicine and Surgery, Minneapolis, 1903; aged 49; died, December 2, of cerebral hemorrhage.

Clarence L. Mershon ✠ Fairhope, Ala.; Drake University College of Medicine, Des Moines, 1898; past president of the Baldwin County Medical Society; aged 54; died, November 26.

John Alexander Cox, Donaldson, Ark.; Memphis Hospital Medical College, 1889; member of the Arkansas Medical Society; aged 53; died, November 29, at a hospital in Little Rock.

George W. Schwartz, Summer Hill, Ill.; St. Louis Medical College, 1873; member of the Illinois State Medical Society; aged 78; died suddenly in November, of cerebral hemorrhage.

Edward B. Mouser, Electra, Texas; University of Louisville (Ky.) School of Medicine, 1898; at one time mayor of Electra; aged 50; died, December 2.

Edward Harris Scatchard, Sewickley, Pa.; Hahnemann Medical College and Hospital of Philadelphia, 1901; aged 49; was found dead, December 1.

Joseph Leo Gavin, Boston (licensed, Massachusetts, 1898); aged 55; died, November 29, at the Peter Bent Brigham Hospital, of heart disease.

James O. McDowell, Sumner, Ill.; Eclectic Medical Institute, Cincinnati, 1874; aged 82; died, November 28, at the Olney (Ill.) Sanatorium.

Maximillian Charles von Groll, Boston; Tufts College Medical School, Boston, 1898; aged 52; died, November 11, of chronic myocarditis.

John W. Wilhoit, St. George, Kan.; Northwestern Medical College, St. Joseph, 1885; formerly county coroner; aged 72; died, November 28.

William M. Durham, Jr., Woodville, Ga.; Georgia College of Eclectic Medicine and Surgery, Atlanta, 1906; aged 42; died, November 29.

Almarian W. Dyer, Butler, Ill.; Starling Medical College, Columbus, Ohio, 1883; aged 72; died, December 2, of carcinoma.

Benjamin F. Holliday, Atlanta, Ga.; Southern Medical College, Atlanta, 1894; aged 59; died, November 29, at a local hospital.

Parker T. Trowbridge ✠ Hayward, Wis.; Milwaukee Medical College, 1910; aged 38; died, in November, of heart disease.

Thomas W. Harris, Eugene, Ore.; University of California Medical School, San Francisco, 1875; aged 77; died, November 30.

Oliver B. Richards, Nova, Ohio; University of Wooster Medical Department, Cleveland, 1874; aged 76; died, December 3.

Charles Edward Page, Melrose Highlands, Mass. (years of practice); Civil War veteran; aged 85; died, November 23.

Louis Feldman, Brooklyn; Long Island College Hospital, Brooklyn, 1910; aged 46; died, December 3, of heart disease.

Elison V. Buckley, Senath, Mo.; Willamette University Medical Department, Salem, 1881; aged 74; died, October 20.

Charles Lee Nollau, Henderson, Ky.; University of Louisville School of Medicine, 1901; aged 45; died, December 3.

Dave C. Sanders, Boswell, Okla. (licensed, Oklahoma, under the Act of 1908); aged 74; died, November 27.

John Leonard Marsh, Brownsburg, Ind.; Medical College of Ohio, Cincinnati, 1874; aged 74; died, December 4.

Elias Kline Boltz ✠ Lebanon, Pa.; Jefferson Medical College of Philadelphia, 1896; aged 54; died, August 19.

Felix G. Thornton, Knightsville, Ind. (licensed, Indiana, 1897); aged 75; died, December 8.

Norman Hull, Howard, Ohio (licensed, years of practice); aged 85; died, November 24.

Correspondence

SYNERGISM OF MAGNESIUM SULPHATE AND MORPHIN

To the Editor:—In THE JOURNAL, November 7, appeared an article by Dr. Gwathmey which he describes as a reply to my work on the alleged synergism of magnesium sulphate and morphin. There are three tables. Table 1 furnishes data of a case in which the patient experienced greater relief from pain when given morphin plus magnesium sulphate than when given morphin alone. This mere fact does not, however, constitute proof that simple addition did not bring about this result; indeed, the second longest period of relief was experienced in one of the two instances only in which magnesium sulphate alone was used. Tables 2 and 3 comprise lists of cases in which the time before postoperative sedative was shown to be longer when morphin plus magnesium sulphate was used preoperatively than when morphin was used alone. Here again there is no evidence of synergism. Both experimental and clinical literature offer abundant proofs that magnesium sulphate is a powerful sedative, which evidence I have never sought to question; in fact, I have published certain observations of the powerful depressing effect it has on dogs. However, that the salt has the ability to raise specifically the sedative power of morphin either in dogs or in man, I do hold to be not proven.

Dr. Gwathmey's discussion of the synergism of magnesium sulphate and ether and his introduction of procain into the magnesium sulphate-morphin mixture serve only to confuse the studies.

HARRY BECKMAN, M.D., Milwaukee.
Acting Director, Department of Pharmacology,
Marquette University School of Medicine.

"SYMMETRICAL CYANODERMA"

To the Editor:—In the Rio de Janeiro letter (THE JOURNAL, November 28) appears the abstract of a report by Dr. Moreira de Fonseca, dealing with a matter entitled symmetrical cyanoderma.

The description of the eruption is meager, but, judging from the brief account, the lesions and their mode of involution are very strongly suggestive of an eruption due to either phenolphthalein or antipyrin, administered by mouth.

It would be unwise to burden our overcrowded dermatologic nomenclature with the term "symmetrical cyanoderma," for it might be readily confused with such diseases as erythromelalgia, Raynaud's disease, pernio and erythredema.

FRED WISE, M.D., New York.

Queries and Minor Notes

ANONYMOUS COMMUNICATIONS and queries on postal cards will not be noticed. Every letter must contain the writer's name and address, but these will be omitted, on request.

AUTOMOBILE ANTIFREEZING SOLUTIONS

To the Editor:—What are the most commonly used antifreeze mixtures and what are their advantages and disadvantages? What are the percentages of ingredients to prevent freezing in this locality? Please omit name.

T. H. P., M.D., Chicago.

To the Editor:—About a year ago I saw an answer to a query in regard to an antifreezing solution for use in winter in our cars. This article I have mislaid and cannot find. Would you be kind enough to send me that formula and inform me as to whether it evaporates, injures a radiator, at what point it boils, at what point it freezes and how strong a solution should be used? Or if you have by this time a better formula, you may send me that one. There are several on the market at this time made from a glycerin base, but they are all expensive. If published, use initials only.

C. P. S.

ANSWER:—There are five types of antifreezing solutions for automobile radiators in use, each of which has certain advantages and disadvantages:

1. Alcohol (denatured). This is in most common use, approximately 28,000,000 gallons having been consumed in the United States for this purpose in 1924-1925. It has the advantages of being readily obtainable, of depressing the freezing point, and of having a satisfactory viscosity. The "strength" of the alcohol-water solution in the radiator can be measured by means of an hydrometer. Its shortcomings are that it has a lower boiling point than water; therefore, if proper precautions are not taken it will boil over; the solution loses its nonfreezing properties through continued use by evaporation of the alcohol, as a result of the relatively high vapor pressure. The alcohol vapors from overheated radiators are also inflammable, and the denaturants used are often obnoxious. Alcohol attacks automobile finishes.

2. Glycerol (glycerin). Glycerol, which should be of a relatively good grade, has the advantages that, once introduced into the radiator system in aqueous solution, it remains there unless lost through leakage or is removed intentionally; that is, it does not lose its effectiveness from use. Moreover, it is odorless and does not have the solvent action on finishes, as has alcohol. On the other hand, it is somewhat more expensive than alcohol, and as it is a by-product it is doubtful whether production can be increased sufficiently to meet the demand if it replaces alcohol. There has been an erroneous opinion that glycerol attacks the rubber connections. According to H. S. Corth (*Pharmaceutical Era*, Feb. 14, 1925, p. 212), the explanation is that some radiator hose is swelled by water. When this happens, the holding clamps dent into the hose. Glycerol tends to cause the hose to return to its original size, but the dents made by the clamps will remain in the rubber. The obvious cure, of course, is to tighten the clamps.

The following table, worked out by the Bureau of Standards, gives the temperature at which freezing starts, showing glycerol and alcohol to be of about equal value in lowering the freezing point:

Per Cent. by Volume	Glycerol	Denatured Alcohol
10	29 F.	27 F.
20	21	19
30	12	10
40	0	— 2
50	—15	—18

It should be remembered that solutions of both of the foregoing go through a mushy state before they get solid, so that a solid block of ice would not be formed until 10 or 15 degrees F. below the temperature given in the table.

The boiling points are as follows:

Per Cent. by Volume	Glycerol, Boiling Point, ° F.	Per Cent. Glyc. in Vapors	Alcohol, Boiling Point, ° F.	Per Cent. Alcohol by Weight in Vapors
10	214	0	199	49
20	216	0	190	64
30	217	0	187	71
40	221	0	183	76
50	225	0	181	80

3. Glycerol and Alcohol Mixtures: These are more satisfactory than alcohol alone because there is less loss of alcohol by evaporation; they are less expensive than glycerol alone because of the less amount required. To a certain degree they have some of the disadvantages of both alcohol and glycerol; one drawback is that the "strength" of the solution cannot be tested by an hydrometer because it contains three components.

The following table, taken from the *Pharmaceutical Era* (Dec. 6, 1924, p. 595) gives the freezing temperatures of alcohol-glycerol mixtures as:

Alcohol	Glycerol	Water	Freezing Point
15%	15%	70%	zero
16%	16%	68%	5 below
20%	20%	60%	20 below

4. Glucose or Honey Mixtures; Calcium Chlorid Mixtures: Glucose has been reported with success by a number, while others have found that it becomes "slushy" when the temperature is near zero, or that it causes overheated engines. It was thoroughly discussed in THE JOURNAL last year (Oct. 25, 1924, p. 1356; Dec. 13, 1924, p. 1942; Dec. 13, 1924, p. 1943; Dec. 27, 1924, p. 2113).

Calcium chlorid has also been used, but has generally been discarded because of its action on the metal.

5. Miscellaneous; Kerosene and Ethylene Glycol: Kerosene can be used with advantage in climates where the weather is severe. In warmer climates it causes a greatly overheated engine on warm days because of its poor properties of heat transfer. As there is no vaporizable component, the motor meter probably would not indicate high temperatures in the radiator system.

A new substance, ethylene glycol, has been proposed. From the research work done, it has promise of being of considerable merit (Ethylene Glycol; Curme, George, *Indust. & Engin. Chem.*, November, 1925, p. 1117). It possesses the advantages of alcohol and glycerol combined. It does not act on metals, is permanent, and has physical properties that permit maintenance of optimal engine temperature. It is not yet available in many localities, but large quantity production is promised. It requires a little less amount (by volume) than denatured alcohol for the same freezing points.

TRANSPLANTATION OF THE CORNEA

To the Editor:—Has there been reported a case of transplantation of the cornea? Please help me go over the literature. I will be much obliged for the information. I happen to think of this because I know of a friend who had a traumatic opacity of the cornea of his right eye, and all hopes were given up for recovery. If skin could be transplanted and grow, why could not the same thing happen with corneal transplant?

R. T. ALTURA, M.D., Detroit.

ANSWER.—Much work has been done with regard to the transplantation of corneal tissue to replace scarred cornea. A bibliography of all important articles to 1921 is appended, the most important ones being starred. Of all of the keratoplastic operations, the von Hippel through and through trephine operation has proved the most successful, particularly in the hands of Elschnig and his assistants. The latest report from that clinic concerned 115 cases in which corneal transplantation had been done. Economically useful vision was restored in approximately 10 per cent. of the cases; no change in vision occurred in about 65 per cent.; the vision became worse in about 15 per cent., and the eye was lost in about 10 per cent. The factors determining the success or failure of the operation could not be determined (apart from lack of strict asepsis), and the end-result did not become apparent until about six months had elapsed after operation.

Ascher: *Arch. f. Ophth.** **89:** 339.
Bonnefon and Lacoste: *J. de méd. de Bordeaux* **4:** 11, 1912.
Burke, J. W.: *Arch. Ophth.* **50:** 147 (March) 1921.
Czermak-Elschnig: Augenärztliche Operationen, Ed. 2, **2.**
Elschnig: Prag. med. Wchnschr. **39,** No. 30, 1914; *Naturw. Kl. d. k. Akad. d. Wissensch.*, Vienna **8:** 3, 1911; *Heidelberger* Berichte,* 1920; *Klin. Monatsbl. f. Augenh.*, February, 1914; Ulbrich, *Arch. f. Opht.*, 1909.
Von Hippel: *Arch. f. Ophth.*, 1888, p. 108; **22:** 79; **24:** 335.
Kraupa: *Centralbl. f. prakt. Augenh.*, 1914, p. 132; *Arch. f. Ophth.* **80,** 489.
Löhlein: *Arch. f. Augenh.* **67:** 398, 1910.
Magitot: Paris Ophthamological Society,* June 3, 1913.
Mühlbauer: *Schmidt's Jahresbericht* **35:** 267.
Plange: *Klin. Monatsbl. f. Augenh.*, 1912, p. 490.
Salzer: *Heidelberger Berichte*, 1917.
Szimanowski: *Klin. Monatsbl. f. Augenh.*, 1913, p. 870.
Wagenmann: *Arch. f. Ophth.* **34:** 217, 1888.
Walker, S., Jr.: *Ophth. Rec.* **26:** 383 (Aug.) 1917.
Wiener, Meyer: Regeneration of the Cornea,* THE JOURNAL, Sept. 4, 1909, p. 762.
Gradle, H. S.: Am. J. Ophth.* **4:** 895 (Dec.) 1921.

SCHNABEL'S METHOD OF OCULAR DIAGNOSIS

To the Editor:—Can you give me any information concerning the methods of Dr. Rudolph Schnabel of Munich, in his examination of the eye? I am informed that he makes many extraordinary claims as to the information which he can gain through an examination of the eye with his iridoscope. Some remarkable photographs of the human eye, magnified many times, were recently shown to me by one of his co-workers. My impression is that there is much truth in many of his assertions, but that many of his deductions are unjustified. Any information which you can supply me concerning this subject will be much appreciated.

J. J. SHORT, M.D., New York.

ANSWER.—A letter from Munich in the *Deutsche medizinische Wochenschrift* states that the Munich Aerztliche Verein recently devoted a session to "Homeopathy and Eye-Diagnosis," as both are much the fashion there at present and popular among the public. In the center of general interest is diagnosis by inspection of the eye, which is practiced partly by registered physicians, partly by laymen, partly

with all modern equipment of apparatus, partly with the simple ophthalmoscope, but always with much success, according to the one making the diagnosis. Dr. Salzer, the professor of ophthalmoscopy, read the paper on this "So-Called Eye Diagnosis," and, the author remarks, "die wissenschaftliche Wertlosigkeit dieser Methode trat damit klar zutage" (the scientific worthlessness of this method was made very plain by what he said). . . . "Professor Salzer invited an eye-diagnosis colleague to a public test of his method, but the invitation has not been accepted."

DISTRIBUTION OF WHITE AND COLORED PHYSICIANS

To the Editor:—Would it be possible for you to get for us through your office, or tell us where we could get, the number of male white physicians in the United States in 1924, by States, and for the following cities: New York, Chicago, Philadelphia, Los Angeles, Cleveland, Boston, Detroit, Baltimore, St. Louis and Pittsburgh? We need these figures for the study of deaths among physicians by age groups and would be grateful if you could help us out.

HARRIET HUGHES, New York.

ANSWER.—The following table lists as nearly as possible the number of white and colored physicians in each state:

	Colored	Male and Female, White (Estimated)
Alabama	163	2,121
Arizona	...	378
Arkansas	95	2,117
California	25	8,338
Colorado	6	1,831
Connecticut	9	1,875
Delaware	17	239
District of Columbia	150	1,663
Florida	94	1,358
Georgia	255	2,867
Idaho	...	416
Illinois	132	10,611
Indiana	30	4,221
Iowa	5	3,373
Kansas	40	2,324
Kentucky	153	2,888
Louisiana	134	1,857
Maine	...	1,037
Maryland	73	2,240
Massachusetts	59	6,128
Michigan	42	4,795
Minnesota	12	2,811
Mississippi	107	1,595
Missouri	152	5,654
Montana	...	525
Nebraska	11	1,858
Nevada	...	129
New Hampshire	3	598
New Jersey	99	3,468
New Mexico	2	363
New York	175	17,496
North Carolina	254	2,027
North Dakota	...	285
Ohio	77	7,036
Oklahoma	180	2,344
Oregon	...	1,176
Pennsylvania	188	10,952
Rhode Island	11	760
South Carolina	153	1,164
South Dakota	...	604
Tennessee	154	2,974
Texas	180	5,883
Utah	...	505
Vermont	...	537
Virginia	200	2,334
Washington	3	1,778
West Virginia	69	1,684
Wisconsin	2	2,824
Wyoming	...	255
Total	3,514	143,496

ACNE ROSACEA

To the Editor:—Please give me information concerning the treatment of acne rosacea, including the administration of the endocrine gland products. Please omit my name.

T. J. R., Kentucky.

ANSWER.—Several factors have to be considered in the treatment of acne rosacea. Errors of diet and gastrointestinal disturbances of other sorts are contributing factors, and an examination has to be made to determine the presence or absence of these and treatment instituted according to the findings. Occasionally desiccated thyroid (thyroideum siccum U. S. P.) is of service in women at the climacteric period. This should not be given, however, without determining its propriety by having a basal metabolism test made. Local treatment is of service but has to be regulated according to the character of the symptoms present. These are variable in different cases.

Medical Education, Registration and Hospital Service

COMING EXAMINATIONS

ALABAMA: Montgomery, Jan. 12. Chairman, Dr. S. W. Welch, Montgomery.

ARIZONA: Phoenix, Jan. 5-6. Sec., Dr. W. O. Sweek, 404 Heard Bldg., Phoenix.

COLORADO: Denver, Jan. 5. Sec., Dr. David A. Strickler, 612 Empire Bldg., Denver.

DISTRICT OF COLUMBIA: Jan. 12. Sec., Dr. Edgar P. Copeland, Apt. 104, Stoneleigh Court, Washington, D. C.

HAWAII: Honolulu, Jan. 11. Sec., Dr. G. C. Milnor, 401 So. Beretania St., Honolulu.

ILLINOIS: Chicago, Jan. 19-21. Supt. of Reg., Mr. V. C. Michels, Springfield, Ill.

INDIANA: Indianapolis, Jan. 12. Sec., Dr. W. T. Gott, 421 State House, Indianapolis.

MINNESOTA: Minneapolis, Jan. 5-7. Sec., Dr. Thomas S. McDavitt, 734 Lowry Bldg., St. Paul.

NEW YORK: Albany, Buffalo, New York, Syracuse, Jan. 25-28. Chief, Mr. Herbert J. Hamilton, Department of Education, Albany.

NORTH DAKOTA: Grand Forks, Jan. 5. Sec., Dr. George M. Williamson, Grand Forks.

OREGON: Portland, Jan. 5-7. Sec., Dr. N. C. Coe, 1208 Stevens Bldg., Portland.

PENNSYLVANIA: Philadelphia, Jan. 26-30. Act. Sec., Mr. C. D. Koch, Harrisburg.

RHODE ISLAND: Providence, Jan. 7. Sec., Dr. Byron U. Richards, State House, Providence.

SOUTH DAKOTA: Pierre, Jan. 19. Dir. Med. Licensure, Dr. H. R. Kenaston, Bonesteel, S. D.

UTAH: Salt Lake City, Jan. 5. Director of Reg., Mr. J. T. Hammond, 412 State Capitol Bldg., Salt Lake City.

WASHINGTON: Olympia, Jan. 12. Sec., Mr. William Melville, 15 Insurance Bldg., Olympia.

WEST VIRGINIA: Charleston, Jan. 19. Sec., Dr. W. T. Henshaw, State Health Department, Charleston.

WISCONSIN: Madison, Jan. 12-14. Sec., Dr. Robert E. Flynn, 315 State Bank Bldg., La Crosse.

Pennsylvania July Examination

Mr. C. D. Koch, director of professional education, Bureau of the Pennsylvania Board of Medical Education and Licensure, reports the oral, written and practical examination held at Philadelphia and Pittsburgh, July 7-11, 1925. The examination covered 5 subjects and included 50 questions. An average of 75 per cent. was required to pass. Of the 289 candidates examined, 273 passed, and 16 failed. Eight candidates were licensed by reciprocity, and 4 by endorsement of credentials. The following colleges were represented:

College	PASSED	Year Grad.	Number Passed
Georgetown University School of Medicine		(1924)	2
George Washington University Medical School		(1924)	1
Howard University School of Medicine		(1924)	2
Northwestern University Medical School		(1925)	3
University of Illinois College of Medicine		(1925)	2
Indiana University School of Medicine		(1924)	1
Tulane University of Louisiana School of Medicine		(1922)	1
Johns Hopkins University Medical Department		(1923)	1
University of Maryland School of Medicine	(1923), (1924, 4)		5
Harvard University		(1924)	1
Tufts College Medical School		(1924)	2
University of Michigan Med. School	(1887), (1921), (1924, 2)		4
St. Louis University School of Medicine		(1924)	1
University of Nebraska College of Medicine		(1923)	1
Cornell University Medical College		(1924)	2
New York Medical College and Hospital for Women		(1905)	1
University and Bellevue Hospital Medical College		(1924)	1
University of Buffalo Department of Med.	(1914), (1924, 2)		3
Eclectic Medical College, Cincinnati		(1924)	2
Ohio State University College of Medicine		(1923)	1
University of Cincinnati College of Medicine		(1924)	1
Hahnemann Med. College and Hospital of Philadelphia		(1924)	31
Jefferson Medical College of Philadelphia	(1908), (1922), (1923, 12), (1924, 65)		79
Temple University Department of Med.	(1923), (1924, 12)		13
University of Pennsylvania School of Medicine	(1919), (1922, 2), (1923, 6), (1924, 36)		45
University of Pittsburgh School of Medicine		(1924)	30
Woman's Medical College of Pennsylvania		(1924)	5
Meharry Medical College		(1916)	1
University of Texas Department of Medicine		(1923)	1
Medical College of Virginia	(1919), (1923), (1924)		3
University College of Medicine, Richmond		(1899)	1
Queen's University Faculty of Medicine		(1924)	1
McGill University Faculty of Medicine		(1924)	1
University of Toronto Faculty of Medicine	(1916), (1923, 4), (1924, 11)		16
Comenian Univ., Bratislava, Czechoslovakia	(1919),* (1921)*		2
Licentiate of the Royal College of Physicians and Surgeons, London, England		(1923)	1
University of London, England		(1923)	1
University of Berlin, Germany		(1923)*	1
University of Heidelberg, Germany		(1921)*	1
University of Munich, Germany		(1921)*	1
University of Zurich, Switzerland		(1917)	1

College	FAILED	Year Grad.	Number Failed
Howard University School of Medicine		(1924)	2
Kentucky School of Medicine of Louisville		(1903)	1
Maryland College of Eclectic Medicine and Surgery		(1913)	1
Temple University Department of Medicine		(1922)	1
University of Pittsburgh School of Medicine		(1924)	1
Woman's Medical College of Pennsylvania		(1924)	2
University of Tennessee College of Medicine		(1909)	1
Vanderbilt University School of Medicine		(1923)	1
German University of Prague, Czechoslavakia		(1922)*	1
University of Marburg, Germany		(1923)*	1
University of Budapest, Hungary		(1906)*	1
University of Naples, Italy		(1916),* (1921)*	2
University of Rome, Italy		(1910)*	1

College	LICENSED BY RECIPROCITY	Year Grad.	Reciprocity with
Howard University School of Medicine		(1915)	Missouri
Northwestern University Medical School		(1914)	Wisconsin
Indiana University School of Medicine		(1920)	Indiana
University of Michigan Medical School		(1917)	Michigan
College of Phys. and Surgs. in the City of New York		(1895)	New York
Jefferson Medical College of Philadelphia		(1912) New Jersey, (1921)	R. Island
University of Pennsylvania School of Medicine		(1921)	N. Carolina

College	ENDORSEMENT OF CREDENTIALS	Grad. Year	with Endorsement
University of Pennsylvania School of Med.		(1922), (1923)	N. B. M. Ex.
Woman's Medical College of Philadelphia		(1923, 2)	N. B. M. Ex.

* Graduation not directly verified by the American Medical Association.

Book Notices

THE ANATOMY OF THE ROOT-CANALS OF THE TEETH OF THE PERMANENT DENTITION. By Walter Hess, M.D., D.D.S., Professor of Operative Dentistry in the Dental College of the University of Zürich. The Anatomy of the Root-Canals of the Teeth of the Deciduous Dentition, and of the First Permanent Molars. By Dr. Med. Dent. Ernst Zürcher, School-Dentist of Zürich. Cloth. Price, $5.50. Pp. 199, with illustrations. New York: William Wood & Co., 1925.

The authors have divided their book into two main parts, the first dealing with the anatomy of the root-canals of the permanent teeth. The first author gives a thorough review of the previous literature concerning the subject and then describes in detail the technic used in his own researches. Each tooth of the permanent set is given a separate chapter in which the minute anatomy of its root-canal is described, with a tabulation of the number of teeth examined, the number of canals found, the apical ramifications, the marrow canals, and the average percentage of each. In making this study the author has examined 2,800 teeth in persons ranging in age from 5 to 55 years. This part of the book is profusely illustrated, and credit should be given the publishers for their careful treatment of the illustrations. In the second part, the anatomy of the root-canals of the deciduous teeth, the author's treatment has been the same, and there are many beautiful illustrations. Since the proper treatment of disease of the dental pulp and of canals is based on the anatomy of the parts, every dentist who attempts such treatment should be thoroughly familiar with this excellent work. The anatomy of dental pulp is a most important factor in the problem of the pulpless tooth and its relation to systemic disease. All interested in this subject will find valuable material for study in this volume.

THE NURSING OF EYE CASES. By Louise Kingham, S.R.N., Matron of the Weymouth and Dorset County Royal Eye Infirmary. Paper. Price, 30 cents. Pp. 16. New York: Oxford University Press.

There can be no question in the mind of the reader of this pamphlet that Miss Kingham is an efficient and capable ophthalmic nurse; but she does not tell any one else how to be. The anatomy of the eye is described more briefly and in a more elementary manner than is usual in a grammar school physiology, although words of more than one syllable are used. Some of the commoner diseases of the eye are mentioned, and the treatment of each is spoken of briefly. But the treatment described is that employed by the surgeon, while the nurse is warned that she must not "even put drops in an eye without an order from the surgeon." The essentials of the technic of ophthalmic nursing are carefully hidden away in the text, without any cohesion. The illustrations so necessary for this type of instruction are lacking, and the word pictures that might take their place are stilted in style. We may demand too much from our nurses in this country, but certain it is that an American nurse could not go far on what she might learn from this pamphlet. On the other hand, instructions are contained in this book for the dressing of patients after operation for cataract, a procedure that is always carried out here by the surgeon or one of his medical assistants. Taken by and large, it is difficult to see any great sphere of usefulness for this book in this country.

ALLERGY, ASTHMA, HAY FEVER, URTICARIA AND ALLIED MANIFESTATIONS OF REACTION. By William W. Duke, Ph.B., M.D. Cloth. Price, $5.50. Pp. 339, with 75 illustrations. St. Louis: C. V. Mosby Company, 1925.

The author concerns himself only with the clinical side of the subject, using chiefly quotations for its purely theoretical aspects. This feature, frankly admitted by the author, constitutes the weakness and strength of the book. The author utilizes a great deal of practical experience in a disease which he finds to some degree present in as many as 15 per cent. of subjects. This is important, since the manifestations of allergy are apt to produce particularly disagreeable symptoms and are accessible to treatment, if interpreted correctly. Unfortunately, too much stress is put on too exact diagnosis, which the average patient cannot afford, and too little on nonspecific treatment, which is and will have to remain in the hands of general practitioners. Nonspecific treatment is essential for immediate relief, as well as for those cases that yield to it. The specific tests may be negative because of inherent defects of biologic tests or because it is impossible to test for everything. A better outline of treatment, with emphasis on the nonspecific agents, would be valuable. A chapter on anaphylactoid reactions caused by physical agents makes an original and interesting contribution to the subject.

THE NORMAL DIET. A Simple Statement of the Fundamental Principles of Diet for the Mutual Use of Physicians and Patients. By W. D. Sansum, M.S., M.D., Director of the Potter Metabolic Clinic, Department of Metabolism, Santa Barbara Cottage Hospital. Cloth. Price, $1.50. Pp. 72, with 1 illustration. St. Louis: C. V. Mosby Company, 1925.

This, as the subtitle indicates, is a statement of the fundamental principles of diet for the mutual use of physicians and patients. As a statement of principles for physicians, it leaves nothing to be desired; it is interestingly written and easily read. We fail to see how it can be of great service to patients. They cannot well separate principles from practice; for them a book on normal diet should contain instructions on how to construct such a diet. A simple statement of principles is of little practical assistance to the patient.

SONDERBAND ZU "STRAHLENTHERAPIE." Herausgegeben von Prof. Dr. W. Fälta, Prof. Dr. C. J. Gauss, Prof. Dr. Hans Mayer und Prof. Dr. R. Werner. Band IX. Physikalische und technische Grundlagen der Röntgentherapie. Von Dipl.-Ing. Dr. Phil. G. Grossmann. Paper. Price, 13.50 marks. Pp. 300, with 207 illustrations. Berlin: Urban & Schwarzenberg, 1925.

This is devoted to a discussion of the application of roentgen rays to therapeutic purposes. In order to supply the demands of the roentgen-ray therapeutist, as well as those of the scientific investigator, all the accepted theories and their influence on the practice of roentgen-ray therapy are thoroughly discussed according to the best and latest information obtainable. Although some of the intricacies of theory may be beyond the understanding of the practicing roentgenologist, he is supplied at the end of each chapter with a summary of all the practical deductions. Particular stress is laid on the explanation of the theory concerning the absorption of the energy of the roentgen rays. The production of roentgen rays and all the subsequent phenomena are explained by the basic fundamentals of the electron-ether theories, the transformation of the bulk of electronic motion into caloric energy incidentally being mentioned. The concepts of wave length, tension within the tube and the periodicity of the roentgen-ray oscillations also are thoroughly explained. A special chapter is devoted to the discussion of filtration of roentgen rays. In dealing with the biologic action of the rays on living tissues, the author calls attention

to the fact that one must distinguish between immediate and remote affects. The chapter dealing with dosimetric problems is particularly instructive, stress being laid on the principal points governing the construction of the various tubes and apparatus. The usefulness of this chapter is enhanced by a wealth of practical suggestions, covering not only all the details of employment but also the methods of preserving a stable function and the correction of occasionally occurring deficiencies. The protection of the patient and the operator is clearly defined. This book is a reliable guide for the practicing roentgenologist and a valuable reference book for the investigating and constructing electrician.

PARASITOLOGY FOR MEDICAL STUDENTS. By Alex. Mills Kennedy, M.D., Professor of Medicine, University of Wales. Cloth. Price, $3. Pp. 142, with 62 illustrations. New York: Oxford University Press, 1925.

Within brief compass, the author has sought to present to medical students the essential features of parasitology for use in clinical study. One short chapter is devoted to each of the major groups of animal and plant parasites. The introductory matter in these chapters is hardly well put and might properly be omitted, as every one today has available some good textbook on zoology. A marked lack of balance appears in the selection of material. Thus, among flukes *Fasciola hepatica* is given more than two pages, whereas *Clonorchis sinensis* gets only brief mention, and *Paragonimus* and *Fasciolopsis* are not mentioned. The first named species is rare as a human parasite, whereas the others are all of first importance. The only strongyle mentioned is the hookworm, though several others are of real if not equal significance. Among the protozoa also, important forms are omitted, whereas others are treated over fully. There is no excuse today for using such names as *Distomum sinense* or *Taenia mediocanellata*. The colored plate is very successful, and the illustrations of insects are good. Most of those on parasitic worms are poor. The idea of the author, and the form in which it is presented, is good, but unfortunately the execution of the plan is much less successful. Perhaps there may be a reason in an English publication for giving awkward fractions, such as 1/6 and 1/25 inch, and yet one wonders how many microscopists today really can use them.

MOTHER'S MANUAL. The Coming and Care of the Baby. By Dorothy Bocker, A.M., M.D., Instructor of Physical Education at Columbia University. Cloth. Price, $2. Pp. 247, with illustrations. New York: Brentano's, 1925.

This little volume contains the usual amount of information to guide young mothers through their pregnancy and to direct them in the care of the infant. The book is well illustrated, and there is even a diagram entitled "Spanking the Baby." This is intended, of course, to illustrate a method of resuscitation immediately after birth. One may well imagine that a great deal of time and effort was spent in preparing the anatomic and physiologic portions of this book, though it really seems time wasted to attempt to inform the busy mother about these subjects. What she needs is dogmatic directions about the care, feeding, bathing and airing of her baby. We are continually surprised by the fecundity of this variety of book. Would that it were possible for some legislative body or national academy to take a census of the actual number of these books already in existence in the English language, so that a new society for the regulation of birth control might limit the ever increasing supply of them.

A SHORT KEY TO BOTH SEXES OF THE ANOPHELINE SPECIES OF INDIA, CEYLON AND MALAYA. By C. Strickland, M.A., B.Ch., Professor of Medical Entomology in the School of Tropical Medicine, Calcutta. Boards. Price, Re. 1/12. Pp. 19, with illustrations. Calcutta: Thacker, Spink & Co., 1925.

The importance of identifying mosquitoes correctly is so evident in public health work that all aids will be welcomed in a task that at best is difficult. Strickland's key is admirable. The book is concise, well arranged and illustrated with great skill. One rarely finds so successful an effort to place highly technical data at the command of those who need it but are not trained in the particular field.

Medicolegal

Practicing Medicine—Treatment of Disease—Evidence

(Wideman v. State (Ala.), 104 So. R. 438. Ex parte Wideman (Ala.), 104, So. R. 440)

The Court of Appeals of Alabama holds, in *Wideman* v. *State*, that it was not necessary to aver that the defendant (Wideman) treated human diseases by a "system of treatment." It was sufficient to aver that he treated or offered to treat diseases of human beings without first having obtained a license from the state board of medical examiners. One who treats, or offers to treat, diseases of human beings by the system known as "chiropractic" or as "osteopathy" or by any system of medicine in any of its branches or departments, as a profession and means of livelihood, without first having obtained a certificate of qualification from the state board of medical examiners, is "practicing medicine" within the meaning of the statute, and may be found guilty of "practicing medicine without having first obtained a certificate of qualification, etc." While each treatment constitutes a separate offense, numerous treatments by one holding himself out to the public as a practitioner of chiropractic constitute "practicing medicine" as those words are construed in *Bragg* v. *State*, 134 Ala. 165, 32 So. 767. Evidence of many treatments of the same person was permissible under a count of an indictment charging "practicing medicine." But the state may be required to elect for which treatment it prosecutes under a count of an indictment charging that the defendant did treat, or offer to treat, diseases of human beings without having obtained a certificate of qualification from the state board of medical examiners. Under such count, evidence of more than one treatment is admissible, if limited to the purpose of showing the guilty knowledge of the defendant in administering the treatment for which the state elects to prosecute. It was competent for the state to show that the defendant had offices in Montgomery and had a sign "Chiropractor" over the door. It was also competent for the state to show that the defendant treated the prosecuting witness "for sickness." But it was not competent for the state to prove, over the defendant's objection, that the prosecuting witness paid the defendant for the treatments administered to her by him or what the defendant's charges for such treatments were, unless this was shown to be of the res gestae (essential circumstances) of the treatment. Admitting, however, that technical error was committed in this last respect, still, as the state was, on the undisputed evidence, entitled to a conviction and the penalty imposed was the smallest fine provided by the statute, no injury could possibly have come to the defendant's cause by reason of the admission of evidence that the prosecuting witness paid the defendant for the treatments administered to her by him or what his charges for such treatments were, and the judgment of conviction of practicing medicine without a certificate of qualification is affirmed.

In *ex parte Wideman*, the Supreme Court of Alabama, in denying, on rehearing, a writ of certiorari to review the judgment of the court of appeals, says that it thinks it quite clear that the language of the present statute punishing "any person who treats or offers to treat diseases" was advisedly substituted for the language of the former statute punishing "any person who practices medicine or surgery," etc., in the light of the decision in *Bragg* v. *State*, the purpose being, by the use of a comprehensive and well understood phrase, to foreclose such evasive contentions as the one made in that case. The treatment of disease comprehends diagnosis and the prescription or application of remedies for its alleviation or cure. Besides the allopathic and homeopathic schools of medicine, both of which employ drugs in the treatment of disease, there are a number of drugless systems, such as osteopathy, hydropathy, electrotherapy, thermotherapy, psychotherapy and chiropractic. Manifestly, the phraseology of the present statute is designed to place under the regulation prescribed all those systems for the treatment of human diseases, as well as any other system that may be conceived. The offense denounced is treating, or offering to treat, human

diseases without a certificate of qualification from the state board of medical examiners. The phrase "by any system of treatment whatsoever" is no part of the description of the offense. Certainly we cannot impute to the legislature the utterly irrational and destructive purpose of turning loose on the people of the state a horde of ignorant charlatans and quacks, to practice medicine as they please, so long as they are able to show that they use no system. Such practitioners would be far more dangerous than those who are intelligent and skilful enough to follow some system of treatment. The statute does not prohibit a merely casual and friendly service. The indictment in this case was sufficient without alleging, either generally or specifically, that the defendant used a system of treatment, and a demurrer on that ground was properly held to be without merit.

Sufficient Identification of Roentgenograms

(Greco v. Schmidt (N. J.), 129 Atl. R. 146)

The Court of Errors and Appeals of New Jersey says that in this action to recover for personal injuries to a child run down by a truck the defendant contended that the trial judge had improperly admitted in evidence roentgen-ray plates showing the character of the child's injury. This objection was based on the fact that the physician who produced the plates at the trial had not himself taken them. The plates, however, were taken by another physician, under the supervision of the one who testified, and at a hospital at which the witness was engaged in professional work, and to which the child was taken. The rule is, as laid down in the case of *Robinson* v. *Payne*, 122 Atl. 882, that if the correctness of roentgen-ray photographs is established by the testimony of the person making them, or if they are identified by the surgeon under whose general direction and for whose use they were made, and by whom they were used in his diagnosis of the bodily injury, they are admissible in evidence. This court is therefore of the opinion that the roentgen-ray photographs were properly admitted in evidence, as they were identified by the physician at the hospital to which the child was taken, and were made under his supervision and direction, and for his diagnosis of the injury.

Legal Remedy After Performing Services for Minor

(In re Dzwonkiewicz's Estate (Mich.), 203 N. W. R. 671)

The Supreme Court of Michigan says that a physician performed a surgical operation on, and attended for about five months, a minor who was seriously injured in an automobile accident. The minor's father had abandoned his family. The mother frequently met the physician at the hospital, but the question of compensation was not discussed with her. The probate court appointed her guardian of the minor, and she was paid $6,500, less attorneys' fees, in settlement of the claim for damages which she sought to recover against a company charged with responsibility for the accident. Then the physician filed a petition in the probate court, setting up the foregoing facts, and averring that the services rendered by him for the minor were of the value of $500, and that the guardian had sufficient funds in her hands to pay for such services. He asked for an order requiring payment by her. Proof was taken by the probate court. The charges were found to be reasonable, and an order was entered directing payment thereof by the guardian. That order was reviewed in the circuit court, which set aside the order of the probate court on the ground that the physician had "mistaken his remedy"; that he should have proceeded by suit against the guardian. The supreme court, however, holds otherwise, setting aside the order of the circuit court, and affirming that of the probate court. In doing so, the supreme court says that the services were performed by the physician without express contract for payment. The father had abandoned his family. There was no doubt that the minor was liable for the reasonable value of the services on an implied contract to pay therefor. Such services as were rendered by the physician were "necessaries," within the meaning of that term as applied to the liability of infants to pay therefor. The right of the physician to recover in this proceeding was dependent on the implied contract of the ward to pay him for his services. He had no contract with the guardian. The proceeding to obtain allowance of the claim was akin to that of a creditor with a claim against the estate of a person deceased. There is no more of a denial of the right to a jury trial in such a proceeding than in the allowance of claims against an estate by the probate court. In either case an appeal may be taken to the circuit court, where the liability and the amount thereof may be determined by the jury, if desired.

Power of Minors to Contract for Medical Services

(Aubel v. Sosso et al. (Calif.), 236 Pac. R. 319)

The District Court of Appeal of California, First District, Division 2, says that in this action by a minor suing by his guardian ad litem (for the litigation) to recover damages for injuries caused by being struck by an automobile, the defendants attacked the ruling of the trial court in allowing the question of special damages to be submitted to the jury. This attack was based on the theory that the father of the minor being legally liable to furnish his son with necessary medical attendance, no obligation attached to the son for those expenses and he was not, therefore, entitled to recover for the outlay in this action. It was also argued that if any recovery could be had for the medical attendance furnished to the son, it must be on the suit of the father alone. In support of their position, the defendants cited Sections 35-37 and 207 of the civil code of the state, and from these argued that a minor living with his parents is incapable of entering into a contract even for medical services. No authorities were cited in support of the position taken by the defendants, and from this court's reading of the sections it is satisfied that the civil code did not intend to place such limitations on the power of a minor. This court can see no reason to hold that merely because these sections of the code prohibit a minor from disaffirming a contract for necessaries when not living under the care of parent or guardian, he is thereby disabled from entering into a valid, though voidable, contract for such necessaries while living under the care of a parent or guardian. On the other hand, Section 207 indicates an intention contrary to that suggested by the defendants, because if a third person may, under that section, supply such necessaries and recover the reasonable value thereof from the parent, it is because the minor is legally qualified to contract for such necessaries with the third person. Therefore, without being cited to any authorities which support the propositions, this court is not prepared to hold that a minor may not make such a contract while living under the care of his parent or guardian. If the contract for such necessities on the part of the minor is valid, the fact that the obligation has been met by advancements on the part of the parent prior to the trial of the case is no defense to the recovery of special damages arising out of the defendant's negligence. Judgment for the plaintiff was affirmed, and a new hearing was denied by the Supreme Court of California.

Society Proceedings

COMING MEETINGS

American Pharmacological Society, Cleveland, Dec. 28-31. Dr. E. D. Brown, University of Minnesota, Minneapolis, Minn., Secretary.

American Physiological Society, Cleveland, Dec. 29-31. Dr. W. J. Meek, Science Hall, Madison, Wis., Secretary.

American Society for Experimental Pathology, Cleveland, Dec. 28-30. Dr. E. B. Krumbhaar, Philadelphia General Hospital, Philadelphia, Sec'y.

American Society of Biological Chemistry, Cleveland, Dec. 28-30. Dr. D. W. Wilson, University of Pennsylvania School of Medicine, Philadelphia, Secretary.

Federation of American Societies for Experimental Biology, Cleveland, Dec. 29-31. Dr. Walter J. Meek, Science Hall, Madison, Wis., Sec'y.

Isthmian Canal Zone, Medical Association of, Ancon, Jan. 19. Dr. David L. Robeson, Ancon Hospital, Ancon, Secretary.

Sioux Valley Medical Association, Sioux City, Iowa, Jan. 19-20. Dr. R. F. Bellaire, Trimble Block, Sioux City, Secretary.

Society of American Bacteriologists, Madison, Wis., Dec. 29-31. Dr. James M. Sherman, Cornell University, Ithaca, N. Y., Secretary.

Current Medical Literature

AMERICAN

The Association library lends periodicals to Fellows of the Association and to individual subscribers to THE JOURNAL for a period of three days. No foreign journals are available prior to 1920, nor domestic prior to 1923. Periodicals published by the American Medical Association are not available for lending, but may be supplied on order. Requests should be accompanied by stamps to cover postage (6 cents if one and 12 cents if two periodicals are requested).

Titles marked with an asterisk (*) are abstracted below.

Annals of Surgery, Philadelphia

82: 673-832 (Nov.) 1925

Epithelial Hyperplasia of Breast. G. L. Cheatle, London, Eng.—p. 637.
*Acute Hypotension of Cerebrospinal Fluid Following Cranial Traumatism. E. Stultz and P. Stricker, Strasbourg, France.—p. 678.
End-Results in Neurosurgery. W. Sharpe, New York.—p. 684.
*Surgical Treatment of Epilepsia Partialis Continua (Kojevnickovi). N. J. Beresnegovski, Tomsk, Siberia.—p. 698.
*Periarterial Sympathectomy. I. Cohen, New York.—p. 704.
*Neoplasms of Blood-Lymph-Vascular System; Endotheliomas. D. S. Pulford, Jr., Rochester, Minn.—p. 710.
*Klippel-Feil Syndrome. T. P. Noble and J. M. Frawley, Rochester, Minn.—p. 728.
Horseshoe Kidney. D. N. Eisendrath, F. M. Phifer and H. B. Culver, Chicago.—p. 735.
*Nonparasitic Chyluria. A. A. Kutzmann, Los Angeles.—p. 765.
Mechanism of Acute Osteomyelitis. A. O. Wilensky, New York.—p. 781.

Treatment of Acute Hypotension of Cerebrospinal Fluid.—Stultz and Stricker assert that the rational treatment of acute hypotension of the cerebrospinal fluid is the intravenous injection of from 30 to 40 c.c. of distilled water. The method is at once easy and safe. It may be advisable, in order to secure a definitive result, to repeat the injection twice or more. Sometimes the pressure becomes only stabilized after two or three hypertensive injections. Three cases are cited.

Treatment of Partial Continuous Epilepsy.—This disease is met very often in the north of Siberia, while in Europe it is comparatively rare. Village inhabitants are chiefly attacked with it. Probably the cold climate of Siberia plays some part in the etiology. Young persons, not older than 30, are chiefly affected. Hereditary syphilis is often marked and alcoholism prevails. Recent syphilis usually is not found. The Wassermann test gives negative results. The main symptoms of Kojevnickovi's epilepsy are continual convulsions of particular groups of muscles, as, for example, convulsions confined to one of the limbs. In spite of the strenuous work of the muscles of the affected limb atrophy often occurs. The local convulsions periodically change into total convulsions of epilepsy. The treatments are (1) medical, by the administration of preparations of bromin or mercury, and (2) surgical, as a decompression operation and one for the removal of cortical centers. Beresnegovski reports eight cases.

Periarterial Sympathectomy.—Eleven cases are reported by Cohen. Seven were of arteriosclerotic origin, two were thrombo-angiitic, one was caused by a trophic ulcer, and one by an ulcer in a mild diabetic, perhaps on a thrombo-angiitic basis. Three patients were completely relieved of pain, one of whom, however, lived too short a time to determine the permanency of the relief and one subsequently lost several toes in which dry gangrene existed at the time of the sympathectomy. One of the thrombo-angiitic patients showed slight but only temporary improvement. The trophic ulcer patient was somewhat benefited. In the remaining six cases no benefit could be seen from the operation.

Tumors of Blood-Lymph-Vascular System. — Pulford reviews 200 cases of neoplasm of the blood-lymph-vascular system: 183 angiomas, nine angio-endotheliomas, and eight endotheliomas. The original hypotheses seem established as facts: blood-vascular and lymph-vascular angiomas, while usually benign, are potentially malignant endotheliomas. There is an intermediate stage between these two represented by the angio-endothelioma which is relatively benign but definitely malignant. Malignant endotheliomas of the blood-lymph-vascular system exist as a pathologic entity. Just as a malignant tumor of the skin is recognized as a squamous-cell epithelioma by its differentiation into horny, protective epithelium, so may certain of the blood-lymph-vascular tumors be recognized as endotheliomas by their differentiation into tumor blood vessels.

Klippel-Feil Syndrome.—Numerical reduction of the cervical vertebrae is a comparatively rare congenital anomaly, but undoubtedly is more common than the literature would lead one to believe. Noble and Frawley report two cases. The clinical appearances of patients with this anomaly are characteristic: shortening of the neck, low implantation of the hair and limitation of neck movements. The pathologic changes may vary from absence of one or two cervical vertebrae to complete absence of the whole cervical spine. The condition is not incompatible with long life.

Nonparasitic Chyluria.—A case of unilateral nonparasitic chyluria, associated with a perirenal abscess, is reported by Kutzmann. Treatment is empiric. The intravenous use of neo-arsphenamin and pelvic lavages with from 1 to 3 per cent. silver nitrate solution has given encouraging results. Kidney exploration and nephrectomy have been done in cases showing poor progression.

Illinois Medical Journal, Oak Park

48: 337-420 (Nov.) 1925

Interpretation of Cough Symptoms. F. J. Kalteyer, Philadelphia.—p. 351.
Mental Health; Public Health Problem. C. F. Read, Chicago.—p. 355.
Functions of Skin; Relation to General Medicine. E. F. Mueller, New York.—p. 358.
*Treatment of Septicemias and Intoxications in Infants and Children by Exsanguination-Transfusion. A. Brown, Toronto.—p. 363.
Reclaiming Hopeless Chest Cases. D. W. Deal and H. H. Cole, Springfield.—p. 370.
Progress in Thoracic Surgery. C. A. Hedblom, Madison, Wis.—p. 374.
Gunshot Wound of Heart; Repair Under Local Anesthesia with Complete Recovery. K. A. Meyer and W. A. Brams, Chicago.—p. 379.
Gastro-Enterostomy in Treatment of Duodenal Ulcer. J. A. Hartwell and G. S. Dudley, New York.—p. 383.
Traumatisms of Head. H. C. Mitchell, Carbondale.—p. 391.
Defects in Tone Scale; Relation to Poor Spelling and Loss of Musical Tone. F. L. Alloway, Champaign.—p. 396.
Cholesteatoma Involving Ethmoidal Cells and Antrum of Highmore. G. C. Otrich, Belleville.—p. 397.
Ambulant Treatment for Hernia. G. A. McDonald, Fairfield.—p. 399.
Biology of Ductless Glands. W. L. Shank, DeKalb.—p. 404.
Diagnosis of Exophthalmic Goiter and So-Called Toxic Goiter. W. L. Bowen, Peoria.—p. 408.

Exsanguination-Transfusion in Intestinal Intoxication.—Brown reports on the results obtained from the use of Bruce Robertson's exsanguination-transfusion in the treatment of septicemia and intoxications of 105 cases of acute intestinal intoxications. Fifty-eight were cured; of eighty cases of erysipelas sixty-four were cured. Four cases of malignant scarlet fever were exsanguinated with complete recovery in all. In several cases of septicemia, cure was effected. In fifty-three cases of severe burns, twenty-nine patients were cured. The method has been used in other conditions in which there appeared to exist a form of unknown toxemia and in which all previous methods of treatment were unsuccessful. Instances of improvement and cure were noted in cases of severe acidosis of unknown etiology, primary peritonitis, infectious diarrhea in which there is always an accompanying toxemia, and diabetic coma, while no apparent benefit was noted in cases of uremia, toxemia of intussusception and general peritonitis.

Journal of Nervous and Mental Disease, New York

62: 449-560 (Nov.) 1925

*Brains with Total and Partial Lack of Corpus Callosum; Nature of Longitudinal Callosal Bundle. C. DeLange, Amsterdam.—p. 449.
Schizophrenia; Case. J. H. Cassidy, Washington, D. C.—p. 477.
Studies on Dementia Praecox. Conditions Which Might Have Affected Brain Before Onset of Disease. S. Kure and M. Shimoda, Tokyo.—p. 485.
*Etiology of Hydrocephalus. A. S. Maclaire, New York.—p. 498.

Absence of Corpus Callosum.—DeLange reports the case of a boy without a corpus callosum. The patient was born in asphyxia and did not increase in weight, though he was breast fed. The taking of the bottle in hospital often met with difficulties, because his breathing was oppressed then. The child's case resembled more or less a case of myxedema; he had the hard and harsh voice which is so characteristic of infants suffering from this disease. The forehead was very narrow and retreating; the temporal fossae were both sunken. Inspiration was somewhat difficult, as if there were

an obstacle in the nasopharynx. Heart, lungs and abdomen were normal. The hands were somewhat in pronation and continually moving about (as if playing the piano). The child was slightly hypertonic. After taking the feeding bottle the child often became blue, not on the face, which generally kept its normal color, but on the rest of its head, which assumed a dark blue hue. The number of red corpuscles was 5,800,000; the Wassermann reaction was negative; the fundus oculi was normal. An examination of the skull by roentgen rays was also negative, as was the result of the urine examination. The child died of pneumonia. A detailed postmortem report is given. An extensive polygyria and the absence of the corpus callosum were striking.

Prenatal Cerebral Hemorrhage as Cause of Hydrocephalus.—In view of the fact that a hydrocephalus was present at the second examination eight weeks after birth, together with the absence of any such evidence at birth and an etiologic factor (hemorrhage), possible of producing adhesions, was present at the time of birth, Maclaire holds that it is logical to conclude that the incomplete absorption of the hemorrhage produced the organization residue, and the deposit of this new formed tissue over the cortex and along the base of the brain caused the development of the hydrocephalic condition in the course of eight weeks.

Journal of Pharmacology and Experimental Therapeutics, Baltimore

26: 259-324 (Nov.) 1925

Inactivation of Atropin Sulphate by Rabbit Serum. J. LaBarre, Edinburgh.—p. 259.
*Physiologic Action of Furane. E. M. Koch and M. H. Cahan, Chicago.—p. 281.
*Effect of Sodium Arsenite on Blood Sugar Concentration of Rabbit and Dog. H. B. van Dyke, Edinburgh.—p. 287.
Metabolism of Salicylic Acid. E. G. Holmes, Cambridge, England.—p. 297.
Skin Absorption of Certain Gases. D. C. Walton and M. G. Witherspoon, Washington, D. C.—p. 315.

Action of Furane.—From the experiments made by Koch and Cahan it is evident that while furane has some anesthetic and analgesic properties, its toxicity is so great as to eliminate it from consideration for practical use. It seems to be a general protoplasm poison, completely inhibiting the growth of yeast. When given by inhalation it causes convulsions, apparently due to stimulation of the motor centers in the medulla and cord. This is followed by paralysis of the respiratory center and asphyxia. When administered in small doses by mouth it has a corrosive, solvent effect on the mucosa, causing a copious watery secretion. There is an increased permeability of the blood vessels, frequently producing extensive hemorrhages. Injection of 1.5 c.c. into a 10 kg. dog caused immediate death with symptoms and postmortem changes similar to those seen in acute prussic acid poisoning. There is a marked hyperemic condition of the lungs, dilatation of the veins and a bright, cherry red color of the blood.

Effect of Sodium Arsenite on Blood Sugar.—Van Dyke shows that sodium arsenite, intravenously administered in doses considerably below the lethal, causes a distinct but variable hyperglycemia in the rabbit. Accompanying this hyperglycemia there is a reduction in the blood alkali reserve; there is no significant alteration in blood concentration as indicated by determinations of corpuscle volume or percentage of hemoglobin. The hyperglycemia still appears after bilateral splanchnotomy or right splanchnotomy and left suprarenalectomy. Sodium arsenite, therefore, acts peripherally in all probability by increasing hepatic glycogenolysis. The hyperglycemia may be checked or prevented by insulin. Intravenous injections of sodium arsenite in the dog cause a reduction in the whole blood alkali reserve often with a slight hyperglycemia.

Journal of Radiology, Omaha

6: 431-474 (Nov.) 1925

Electricity in Pelvic Diseases of Women. H. W. Grote, Bloomington, Ill.—p. 436.
End-Results Following Physiotherapy. W. S. Keyting, Salt Lake City.—p. 441.
Treatment of Malignancies by Surgical Diathermy and Roentgen Ray. A. L. Yocom, Jr., Chariton, Iowa.—p. 444.
Skin Reactions to Simultaneous Treatments with Radiant Heat and Soft Roentgen Rays. J. A. Hawkins and H. Clark, New York.—p. 446.
Mediastinal Empyema; Case. W. Shepherd, Rulo, and C. L. Hustead, Falls City.—p. 449.

Kentucky Medical Journal, Bowling Green

23: 501-536 (Nov.) 1925

Acute Inflammatory Tubes. C. W. Hibbett, Louisville.—p. 503.
Acute Appendicitis. B. F. Zimmerman, Louisville.—p. 506.
Gallbladder in an Acute Abdomen. W. E. Fallis, Louisville.—p. 508.
Tubal Pregnancy. G. Aud, Louisville.—p. 510.
Perforations of Stomach and Intestine. I. A. Arnold, Louisville.—p. 512.
Intestinal Obstruction. E. S. Allen, Louisville.—p. 514.
Renal Infection. S. C. McCoy, Louisville.—p. 516.
Muscular Dystrophies. C. E. Gaupin, Louisville.—p. 523.
Juvenile Dystrophies. P. F. Barbour, Louisville.—p. 524.
Orthopedic Considerations of Muscular Dystrophies. J. D. Trawick, Louisville.—p. 526.
Endocrinology of Muscular Dystrophies. E. L. Dravo, Louisville.—p. 527.
Treatment of Muscular Dystrophies. J. J. Moren, Louisville.—p. 528.

Northwest Medicine, Seattle

24: 529-592 (Nov.) 1925

Medicine in Idaho. C. W. Pond, Pocatello.—p. 529.
Achlorhydria Family Tree of Diseases. H. A. Christian, Boston.—p. 531.
Eclampsia. C. J. McCusker, Portland, Ore.—p. 536.
Internist Claims Eclampsia. J. F. Griggs, Tacoma.—p. 539.
Morphin-Scopolamin Analgesia in Obstetrics. A. W. Holman, Portland, Ore.—p. 543.
Gwathmey Method of Synergistic Anesthesia in Obstetrics. C. E. Hunt, Eugene, Ore.—p. 546.
Prolonged Labor. J. T. MacKay, Hillsboro, Ore.—p. 551.
Use of Radium in Certain Types of Uterine Bleeding. O. B. Wight, Portland, Ore.—p. 554.
Management of Summer Diarrhea. P. D. McCornack, Spokane.—p. 556.
Balantidial Dysentery. D. T. Ford, Pullman.—p. 558.
Pellagra, Secondary to Voluntary Inanition. N. W. Jones, Portland, Ore.—p. 559.
Trypanosomiasis. I. P. Bartle, North Bend, Ore.—p. 561.
Treatment of Diabetes. I. C. Brill, Portland, Ore.—p. 562.
Twenty-Four Hours of a General Practice. W. L. Hunt, Klamath Falls, Ore.—p. 564.

Radiology, St. Paul

5: 365-458 (Nov.) 1925

Radiography of Infant Chest; Progression of Chest-Determination of the Normal. W. W. Wasson, Denver.—p. 365.
*Radium and Roentgen Rays in Treatment of Round Cell Sarcoma. B. F. Schreiner and C. C. Herger, Buffalo.—p. 399.
*Granulomas: Hodgkin's Disease, Lymphosarcoma and Leukemia. A. Soiland, Los Angeles.—p. 410.
Roentgen-Ray Diagnosis of Renal Tumors. K. T. Myer, Evansville, Ind.—p. 416.
Radio-Active Substances: Therapeutic Uses and Applications. J. Muir, New York.—p. 425.
Protection from Roentgen-Ray Standpoint. E. C. Jerman, Chicago.—p. 430.
Case of Typhoid Pneumonia. W. W. Beldon, New York.—p. 435.
Case of Detection of Foreign Body. H. L. Harris, Columbus, Ohio.—p. 436.
Simple and Effective Method for Making Positive Film Productions from Roentgen-Ray Negatives. A. R. Sheldon, Highland Park, Ill.—p. 437.
Easel for Rapid Serials in Gastro-Intestinal Examinations. E. G. C. Williams, Danville, Ill.—p. 438.
Roentgen-Ray Consultations of Future. G. Bartlett, Schenectady, N. Y.—p. 439.

Radium and Roentgen-Ray Treatment of Round Cell Sarcoma.—Schreiner and Herger have obtained what is apparently a clinical healing in thirteen out of thirty-five cases of round cell sarcoma which had lasted for periods varying in time up to six years and three months. In one group of cases, irradiation has yielded startling primary results, but recurrencs in other parts of the body and anemias have proved fatal in almost all the cases. In the lymphosarcomatosis cases it is far better to divide a definite amount of irradiation over a period of time, from one to two weeks, in order that the body dose be as small as possible. Roentgen rays and radium together, with arsenic medication, are the methods of choice in the treatment of round cell sarcoma.

Cause of Death in Granulomas.—The terminus of these cases, Soiland says, is nearly always found in the deep abdominal glands where the pathologic change becomes firmly entrenched and resistant to any therapeutic attack. It appears that one may successfully destroy the altered lymph nodes in every part of the economy, even including those of the mediastinum, but the glands in the abdominal cavity are the cause of death.

Southern Medical Journal, Birmingham, Ala.

18: 777-858 (Nov.) 1925

Abscess of Lungs. M. C. Pincoffs, Baltimore.—p. 777.
*Treatment of Pneumonia in Children with Mercurochrome Intravenously. L. D. Hoppe and W. T. Freeman, Atlanta, Ga.—p. 784.
New Nomenclature in Bacteriology. C. Phillips, Richmond, Va.—p. 787.
*Simulation of Brain Lesions by Disease of Paranasal Sinuses. D. D. V. Stuart, Jr., Washington, D. C.—p. 790.
*Sprue in Section of South. W. H. Lewis, Rome, Ga.—p. 794.
Sickle Cell Anemia: Case. S. C. Jamison, New Orleans.—p. 795.
*Milk Sickness. G. R. Wilkinson, Greenville, S. C.—p. 797.
Two Cases of Ichthyosis Treated with Ultraviolet Ray. C. Swanson, Atlanta, Ga.—p. 800.
Report of Two Hip Operations. W. L. Sneed and R. H. Patterson, New York.—p. 803.
Management of Disabling Deformities Following Poliomyelitis. W. B. Owen, Louisville.—p. 808.
Common Nonsurgical Bone Lesions of Childhood. J. A. Foote, Washington, D. C.—p. 812.
Syphilitic Prenatal Treatment. W. J. Young and C. H. Likins, Louisville.—p. 815.
Internist's View of Chronic Disabilities. G. Walsh, Fairfield.—p. 818.
Status of Various Electrical Modalities Used in Diseases of the Ear. L. H. Lanier, Texarkana.—p. 821.
*Screw Worms from Soft Palate. L. S. Gaudet, Natchez, Miss.—p. 824.

Treatment of Pneumonia in Children.—The observations made by Hoppe and Freeman on the effect of mercurochrome-220 soluble on pneumonia are based on a series of forty-six cases, of which twenty-three were bronchial and twenty-three lobar in type. In these forty-six cases, twenty-three patients received mercurochrome and twenty-three were used as controls. Of the twenty-three controls, the average duration of illness was 16.5 days, while with those receiving mercurochrome the average duration of illness was 6.5 days. In three of these, the crisis occurred on or before the third day. The twenty-three patients treated with mercurochrome were sick on an average of 2.25 days after the first dose was given. The mortality of the control group was 39 per cent. Of those treated with mercurochrome, the mortality was 8.5 per cent.

Sinus Disease Simulates Brain Lesions.—Stuart is of the opinion that disease of the posterior ethmoid cells and sphenoidal sinuses at times produces signs and symptoms closely simulating those of organic brain disturbances, especially tumors and epidemic encephalitis. Whether or not this simulation occurs depends on the anatomic relations of the sinuses in question.

Sprue in Section of South.—Lewis' observation has been that there is a large proportion of sprue in the South and that it is not restricted by any degree of latitude. It is probably greatly confused with pellagra and, on account of a definite element of transmissibility, warrants the most careful study. Its principal features involve perverted intestinal digestion, possibly associated with a monilia. This digestive disturbance has, in Lewis' experience, been greatly improved by the feeding of bile salts.

Milk Sickness.—Six cases are reported by Wilkinson. The most striking feature of the clinical picture is the acidosis. The treatment of this single feature, with glucose in the form of molasses, has given prompt relief from the acute intoxication.

Screw Worms in Soft Palate.—In Gaudet's patient, examination of the throat revealed the soft palate to be very much swollen, with a perforation through the central part extending clear through, about the size of a 25 cent piece. This perforation was filled with worms burrowing in every direction into the tissues. Careful examination of the nose showed no worms at all in the nasal cavity at the time, though repeated examinations were made. Sixty-two worms were extracted.

Wisconsin Medical Journal, Milwaukee

24: 288-350 (Nov.) 1925

General Biologic Considerations. M. F. Guyer, Madison.—p. 288.
Biologic Basis of Immunity. K. E. Kassowitz, Milwaukee.—p. 300.
Influence of Ductless Glands on Biologic Reaction. E. C. Kendall, Rochester, Minn.—p. 303.
Cell Reactions in Resistance and Immunity. C. H. Bunting, Madison.—p. 305.
Care of Insane. H. H. Christopherson, Colby.—p 311
Treatment of Finger Infections. R. M. Carter, Green Bay.—p. 312.

FOREIGN

An asterisk (*) before a title indicates that the article is abstracted below. Single case reports and trials of new drugs are usually omitted.

Irish Journal of Medical Science, Dublin

337-400 (Aug.) 1925

*Rotunda Lying-In Hospital Report for 1923-1924. G. Fitzgibbon, R. M. Corbet and N. M. Falkiner.—p. 337.

Maternity Work in Rotunda Lying-In Hospital.—During the year 1923-1924, 1,973 patients were admitted to the Rotunda Lying-In Hospital and 1,703 were confined. Normal vertex was the presentation in 1,498 deliveries. This includes all occipitoposterior positions which underwent normal rotation. Eighteen vertex presentations were finally delivered as persistent occipitoposterior. Face presentation, excluding anencephalus, occurred eight times. Transverse presentation, excluding multiple pregnancies, occurred in five cases. Compound presentations occurred four times. Breech was the primary presentation in fifty-one cases, besides occurring in one of twins on nine occasions. There were twenty cases of twin pregnancies and seventeen cases of prolapse of the cord. Forceps were applied for the delivery of 101 patients, seventy-eight primiparas and twenty-three multiparas. Version was performed thirty-one times. In four cases the fetus was broken up to effect delivery. Persistent primary uterine inertia was present in eight cases. There was only one case of definite contraction ring dystocia. Induction of labor was performed thirty times and induction of abortion or miscarriage seven times. Eight cases of accidental hemorrhage occurred. There were twelve cases of placenta praevia, seven cases of postpartum hemorrhage and three cases of rupture of the uterus. The placenta had to be removed manually nineteen times. There were 163 patients with external measurements considerably under normal. Internal pelvimetry was done only once as dependence was placed far more on the application of the head to the pelvic brim, as made out under anesthesia and by noting the progress of labor, than on the actual dimensions of the pelvis. Delivery was effected by cesarean section nineteen times; six patients had sections done previously; in two patients it was the third section. One patient was delivered by pubiotomy after thirty-six hours in labor, the head being impacted in the brim. In sixteen cases treatment was by induction of labor—eleven times for definite contraction of the pelvis and five for disproportion owing to the large size of the fetus. In 220 patients the urine was found to contain a marked amount of albumin. In three cases hyperemesis occurred. There were ten deaths in the intern department; the causes were: sepsis, rupture of a cesarean scar, rupture of the uterus during evacuation, and hemorrhage in a full term ectopic pregnancy, the patient dying just after laparotomy. One patient died of cardiac failure from myocarditis. In the extern department there were eleven deaths, making in the whole maternity service twenty-one deaths. Twelve patients were admitted on account of pain and were found to have pyelitis. There were three cases of hydatid mole; three cases of multiple fibroids of large size with pregnancy; one case of chorea; four cases of mania and one of marked mental derangement for a few days after eclampsia; one case each of herpes, gallstones, appendicitis, anemia, erysipelas and thrombosis of the saphenous vein.

Medical Journal of Australia, Sydney

2: 471-496 (Oct. 17) 1925

Recent Advances in Cancer Work. A. H. Thwaites.—p. 471.
Diabetes. E. Russell.—p. 479.
*Liver in Umbilical Cord of New-Born Infant; Case. P. L. Hipsley.—p. 484.

Liver in Umbilical Cord.—Hipsley reports the case of an infant seen twelve hours after birth that presented a spherical enlargement of the umbilical cord about 3 inches (7.5 cm.) in diameter in its widest portion. None of it was covered by skin. When this mass was opened, the liver was found within it. The liver was returned within the abdominal cavity. The infant made an uninterrupted recovery, and three weeks later appeared to be perfectly normal.

Archives des Maladies de l'Appareil Digestif, Paris

15: 751-870 (Oct.) 1925

*Pancreatic Fistula. M. Villaret and L. Justin-Besançon.—p. 751.
*Cholesterol in Duodenal Juice. M. Chiray et al.—p. 768.
Duodenal Tube in Diagnosis of Cholecystitis. F. Ramond et al.—p. 778.
Carcinoma of Esophagus and Radium Treatment. Cornioley and Gilbert. —p. 790.
*The Diazo Reactions. G. Piotrowski.—p. 798.

Clinical and Physiologic Study of Pancreatic Fistula.—Villaret and Justin-Besançon have had the opportunity to study for seven months and apply various tests to a fistula of the pancreatic duct, consecutive to a gastro-enterostomy, subtotal gastrectomy and exclusion of the pylorus, in a man, aged 43. The rhythm of the secretion of pancreatic juice appeared markedly changed; the assimilation of nitrogen, phosphorus and sulphur considerably reduced. The internal secretory function of the pancreas appeared unaffected. The pancreatic juice did not contain invertin; trypsin production seemed normal or enhanced. Insulin did not modify the secretion; the influence of atropin was indirect. Injection of magnesium sulphate into the jejunum provoked the appearance of bile, without stimulation of the pancreatic secretion. The influence of hydrochloric acid was the reverse of this. No tendency to a psychic pancreatic secretion could be detected. The fistula did not improve spontaneously, nor heal under the action of insulin or atropin. Only an operation restored the passage of pancreatic juice into the intestine.

Technic for Dosage of Cholesterol in Duodenal Juice.—Chiray and his co-workers describe a simplified Grigaut method for clinical dosage of cholesterol in the duodenal juice, obtained by intubation. With this method, they say, the colorimetric reading is easy and rapid. The findings are sufficiently exact for clinical purposes.

Mechanism of Diazo Reactions.—Piotrowski's study confirmed that the bilirubin in mechanical jaundice is identical with that in hemolytic jaundice. The direct diazo reaction depends on the presence of free bilirubin in the serum; the indirect, on association of bilirubin with the proteins of the plasma. The intensity of adsorption phenomena is connected with the amount of proteins and cholesterol in the blood. Doubt is expressed as to the diagnostic value of the diazo reaction in dubious cases.

Presse Médicale, Paris

33: 1489-1504 (Nov. 11) 1925

*The Blood Pressure in Aviators. R. Cruchet.—p. 1489.

Influence on the Blood Pressure of Variations in Atmospheric Pressure.—Cruchet reiterates that under the low air pressure at high altitudes, the arterial pressure in the flyers increases. The pressure becomes normal and all other phenomena of flight sickness disappear as soon as oxygen is regularly inhaled. He asserts that the influence of oxygen is still manifest at an altitude of 8,000 meters, and probably even higher. Even the untrained can bear without harm an air pressure corresponding to an altitude of 8,000 meters if they inhale oxygen regularly. He gives charts showing the regular rise and fall of the blood pressure above and below normal as the atmospheric pressure in the air chamber was varied to correspond to very high altitudes or sea level.

33: 1505-1520 (Nov. 14) 1925

*Conjugal Neurosyphilis. A. Sézary.—p. 1505.
*Skin Reactivity to Roentgen Rays. L. Neu.—p. 1506.
Gerhardt Urine Test. J. W. Grott.—p. 1507.
Endoscopy of the Abdomen. A. Ravina.—p. 1508.

Pathogenesis of Conjugal Neurosyphilis.—See Paris Letter, page 1902.

Susceptibility of the Skin to Roentgen Rays in Various Diseases.—Neu's investigation indicated that an exaggeration of this susceptibility was most pronounced in exophthalmic goiter; less with tuberculous glands, and still less with fibroma. In exophthalmic goiter, the fact may be partly ascribed to a general irritability of the vessels; in the other affections, neither anemia nor hyperemia of the skin can be incriminated. The factor modifying the skin susceptibility is still a mystery.

Schweizerische medizinische Wochenschrift, Basel

55: 1041-1064 (Nov. 12) 1925

*Cow's Milk and Infants. E. Feer.—p. 1041.
Anemias. Naegeli.—p. 1043.
Climatology of High Altitudes. C. Dorno.—p. 1046.
Endocrine and Cerebral Obesity. A. Oswald.—p. 1048.
Thermal Waters of Baden (Switzerland). J. Markwalder.—p. 1053.
Alveolar Pyorrhea. O. Müller.—p. 1055. C'td.

Cow's Milk and Infants.—The experiences of the last ten years have convinced Feer that the 600 c.c. of cow's milk he had recommended for infants in 1916 is still too much. He does not give more than 500 c.c. daily. He recommends 100 gm. per kilogram of body weight up to the amount mentioned, and adds water, sugar, flour and, after the sixth month, thin gruel, soup, vegetables, fruits, later zwieback and bread crust. Fresh fruit juice (teaspoonful) may be given even as early as in the fourth month. He reduces the milk in the second year to 400 c.c. and later to 200-300 c.c. daily. Sometimes it is better to avoid it entirely, especially with anemia. Two diet lists for children are suggested.

Revista de la Soc. Arg. de Neurología, Buenos Aires

1: 59-81, 1925

*Oneiric Trigeminal Neuralgia. J. M. Obarrio.—p. 59.
Living Cysticercus Racemosus Found in Brain at Decompressive Operation. M. Alurralde and M. J. Sepich.—p. 65.
Acute Delirium with Purulent Stomatitis; Two New Cases. Nerio Rojas and J. C. Belbey.—p. 77.

Nocturnal Trigeminal Neuralgia.—The neuralgia had been constantly recurring for fifteen months, almost exclusively during sleep. Obarrio has found insufficiency of the liver in all such oneiric disturbances, and cures them promptly by a nontoxic diet, supplemented by a glass at each meal of a solution of 10 gm. of sodium sulphate, 5 gm. of sodium phosphate and 5 gm. of sodium bicarbonate in 1 liter of water. The oneiric neuralgia or delirium appears when digestion has reached the peak of absorption from the intestine, with the liver function and the elimination of carbon dioxid at their lowest point. He sometimes gives peptone in addition; this alone cured the nocturnal neuralgia in a case reported in the *Presse médicale* in 1922.

Deutsche medizinische Wochenschrift, Berlin

51: 1893-1934 (Nov. 13) 1925

Scarlet Fever. U. Friedemann and H. Deicher.—p. 1893. C'td.
*Antibacteriophage Action of Human Serum. S. Katsu.—p. 1896.
Tabes and Paralysis in Civilized Peoples. H. Salomon.—p. 1897.
Chronic Arthritis in Palestine. M. Buchmann.—p. 1899.
Ovarian Substitution Treatment. W. Hannes.—p. 1901.
Fractional Examination of the Stomach. L. Bogendörfer.—p. 1902.
Treatment of Placenta Praevia. F. Netzer.—p. 1903.
*Prognosis of Genuine Nephrosis. H. Steinitz.—p. 1906.
Polychromophilic Erythrocytes. G. Volk.—p. 1907.
*Urea in Saliva as Kidney Function Test. Simmel and Küntscher.—p. 1909.
Serologic Reaction with Alcoholic Extracts. Lüttge and v. Mertz.—p. 1911.
Direct Gastroscopy. W. Sternberg.—p. 1911.
Treatment of Gastric Ulcer. Jüngerich.—p. 1912.
Treatment of Itching Dermatoses. W. Joseph.—p. 1915.
Drugs Acting on Blood Vessels. Handovsky and Seyderhelm.—p. 1915.
Social Insurance and Tuberculosis. Blümel.—p. 1916. C'tn.
Action of Minute Amounts. Buttersack.—p. 1928. C'cn.

Antibacteriophage Action of Human Serum.—Katsu found a strictly specific antibacteriophage action in a serum which agglutinated the Flexner type of dysentery bacilli. A similar action was observed in three other serums which also agglutinated these bacilli. Treatment of the serum with the bacilli did not diminish its antibacteriophage action. He agrees with Sonnenschein that the method might prove valuable in diagnosis.

Prognosis of Genuine Nephrosis.—Steinitz reports an instance of complete recovery from grave genuine nephrosis (nephritis with edema without hypertension or hematuria). The boy, 11 years old, recovered under treatment with large doses of urea (up to 100 gm. daily), and is healthy at present, eight years after the disease.

Urea in Saliva as Kidney Function Test.—Simmel and Küntscher confirm the practical value of Hench and Aldrich's salivary urea index.

Klinische Wochenschrift, Berlin

4: 2185-2232 (Nov. 12) 1925

Blood Transfusion in Pediatrics. Opitz.—p. 2185.
*Research on Gastric Secretion. G. Katsch and H. Kalk.—p. 2190.
*Differentiation of Tissues. Christeller and Kaiser.—p. 2193.
*Sleep. R. Heilig and H. Hoff.—p. 2194.
Susceptibility to Tuberculin. G. Brückner.—p. 2198.
Syphilitic Atrophy of Muscle. E. Guttmann.—p. 2200.
*Oxydases of Leukocytes. H. Mielke.—p. 2201.
*Conducting Power of Cerebrospinal Fluid. Schaefer.—p. 2202.
*Function Test of Reticulo-Endothelial System. A. Memmesheimer and G. H. Klövekorn.—p. 2204.
*Stomach Tube and Stimulus. L. Hirschberg and T. Ganskau.—p. 2205.
"Surgical Treatment of Nephritis." M. Zondek.—p. 2206.
Experimental Block of Branch of Bundle of His. D. Scherf.—p. 2207.
*Constancy of Gas Metabolism Under Normal Conditions. M. Reiss and R. Weiss.—p. 2207.
Purulent Foci in Lungs. P. Müller and P. P. Gotthardt.—p. 2208.
Treatment of Syphilis of Aorta. G. Hubert.—p. 2210.
Present Status of Sleeping Sickness. E. Steudel.—p. 2214.
Treatment of Ozena. O. Nast.—p. 2229.

Research on Gastric Secretion.—Katsch and Kalk found in caffein (0.2 gm. of pure caffein in 300 gm. of water) an almost equally good substitute for the alcohol test meal.

Formation of Iron Salt in Differentiation of Tissues.—Christeller and Kaiser treated fresh tissues with iron salts and later with hydrochloric acid and potassium ferrocyanid. The cytoplasm of epithelial cells—especially cancer cells—but also the muscles accepted the iron. They point to a possibility that the presence of lactic acid plays a rôle.

Sleep.—Heilig and Hoff found a "vagotonic" blood pressure reaction to epinephrin during sleep; the alimentary glycemia is higher in normal and paraldehyd sleep. The cerebrospinal fluid contains more sugar and protein, less chlorids. The blood from sleeping persons lowered the number of drops in the Läwen-Trendelenburg frog preparation much less than the blood from the same subjects when awake.

Oxydases of Leukocytes.—Mielke believes that Graham's distinction of four types of neutrophils according to the number of granules containing a peroxidase is based only on technical errors.

Conduction in Cerebrospinal Fluid.—The electric conductivity of cerebrospinal fluids in Schaefer's determinations averaged $1.49 \cdot 10^{-2}$. The extreme figures were $1.05 \cdot 10^{-2}$ and $2.00 \cdot 10^{-2}$.

Function Test of Reticulo-Endothelial System.—Memmesheimer and Klövekorn injected intravenously a fine emulsion of oil, and examined blood smears with the dark field illuminator. The droplets disappeared very slowly in patients with secondary syphilis—especially those under arsphenamin-bismuth treatment. The injection induced a shock in some patients, in whom a rapid disappearance of the fat had been observed.

Stomach Tube and Mechanical Stimulus.—Hirschberg and Ganskau do not believe in the mechanical provocation of gastric secretion. It is only a continuation of the secretion which had been going on before the tube was introduced. It is simply the well known psychic secretion, caused usually by the observation of the breakfast being taken by others in the room. This fasting secretion stops after injection of atropin. A digestive secretion continues in spite of it.

Constancy of Gas Metabolism.—Reiss and Weiss determined the oxygen consumption in mice and rats in twenty-four hour periods. The animals were allowed to move about and eat. The oxygen intake was remarkably constant (± 5 per cent.).

Medizinische Klinik, Berlin

21: 1713-1750 (Nov. 13) 1925

Cardiac Dyspnea. R. Cobet.—p. 1713.
*Suprarenals After Insulin. H. Poll.—p. 1717.
*The Colon Problem. F. Pendl.—p. 1720.
Heating Apparatus for Premature Infants. L. Moll.—p. 1723.
Stenosis of Bronchus. O. Glogauer.—p. 1724.
Serotherapy of Cancer. H. Elsner.—p. 1726.
The Urochromogen Reaction. J. Wirges.—p. 1727.
Hematology of Scarlet Fever. H. Bix.—p. 1728. C'en.
*Treatment of Enuresis. P. Freud.—p. 1729.
Hospitalism of Infants. L. Bytsch.—p. 1730. C'en.
Lability Tests. G. Salus.—p. 1732.
Recent Works on Cardiovascular Disease. E. Edens.—p. 1737.
Science and Economics. E. Loewy-Hattendorf.—p. 1749.

Suprarenals After Insulin.—Poll examined the suprarenal glands in various animals after injections of insulin. He found hyperemia, changes of distribution of lipoids, and loss of chromaffine substance. The latter could be prevented by injections of epinephrin.

The Colon Problem.—Pendl administers at least half a tablespoonful of castor oil for ten days after sutures of the colon. It keeps the contents fluid, and thus avoids the tension of gas behind hard scybala. He lost two out of thirty-seven patients.

Treatment of Enuresis.—Freud applied ethyl chlorid to freeze the skin covering the sacral bone or above the symphysis. He reports excellent results after one or two such procedures. Freezing the chest or back was of no value.

Monatsschrift für Kinderheilkunde, Leipzig

31: 97-192 (Nov.) 1925

Skin Diseases and Infections in Children in 1924. C. Leiner.—p. 97.
Epilepsy of Genital Origin. J. S. Galant.—p. 117.
The Rickets-Tetany Problem. K. Waltner.—p. 129.
*Lactation and Submammary Temperature. L. Moll.—p. 140.
Tonsillitis with Lymphatic Reaction. C. Nelken.—p. 151.
Hyperacidity in Infants. G. Török.—p. 157.
*Complexion in Pyuric Infants. W. Lasch et al.—p. 164.
Reduction Index of Urine. G. Török.—p. 169.
The Idiomuscular Reflex. G. Kán.—p. 174.
Drugs and Alimentary Leukocytosis. G. Török.—p. 178.
Phototherapy of Hydrocele in Infants. G. Török.—p. 181.

Lactation and Submammary Temperature.—Moll observed in lactation a higher temperature in the skin fold below the breast than in the axilla. It is almost as high as the rectal temperature. It is a good indicator of the ability to produce milk.

Complexion in Pyuric Infants.—Lasch, Fischer and Silber found an increased bilirubin concentration in the serum of four of twelve cases of pyuria in infants. These four infants presented the peculiar pale yellowish complexion which is known as a sign of the disease. It is the expression of a septic jaundice.

Münchener medizinische Wochenschrift, Munich

72: 1951-1994 (Nov. 13) 1925

Biochemical Problems. K. Felix.—p. 1951.
Blood Transfusion Technic. R. Stahl.—p. 1952.
*Athletic Training. W. Ewig.—p. 1955.
*Serodiagnosis of Cancer. O. Bott.—p. 1959.
*Hutchinson's Triad and the Endocrines. A. Cedercreutz.—p. 1960.
*Urobilin in Cirrhosis. A. Lichtenstein.—p. 1962.
The Typhoid Group in Bavaria. Rimpau.—p. 1964.
*Sympathicotonus of Brain Arteries in Pregnancy. O. Muck.—p. 1967.
*Liver Function Tests. F. Fischler.—p. 1970. C'en.
Jean Paul F. Richter and Medicine. G. Sticker.—p. 1973.
Treatment of Fresh Tears of Cervix. H. Baumm.—p. 1975.
Public Health Service in Bavaria. Seiffert.—p. 1976.

Athletic Training.—Ewig found a moderate dilatation of the heart in persons training for various sports. This increases the length of the muscle fibers and the energy of their contraction. A hypertrophy was found, instead, in a few cases. An increased amplitude of the blood pressure, and bradycardia were confirmed. Aschner's eyeball compression test, the carotid pressure test, and Erben's test gave about the same results as in persons not under training. Examinations of the blood for changes of the calcium and potassium content revealed hardly any difference, but Chvostek's phenomenon was present in over one half of the athletes (against one third of the controls). The alkali reserve of the blood increased.

Serodiagnosis of Cancer.—Bott confirms the good results with Wigand's tannin test. Yet it is negative with beginning cancer.

Hutchinson's Triad and the Endocrines.—Cedercreutz has collected the evidence which might speak for the possible endocrine genesis of Hutchinson's teeth, parenchymatous keratitis and the typical affection of the labyrinth. The congenital syphilis might attack primarily the endocrine glands.

Urobilin in Cirrhosis of the Liver.—Lichtenstein, using Terwen's method, found a high ratio between the urobilin excreted in the urine and that contained in the feces in cir-

rhosis of the liver. The elimination by way of the urine is also considerable in pernicious anemia or hemolytic jaundice, but the amount in the feces is much larger.

Sympathicotonus of Brain Arteries in Pregnancy.—Muck observed in many pregnant women prolonged white marks on the mucous membrane of the nose when irritating it with a sound after application of epinephrin. He regards this as a sign of increased sympathetic tonus of the arteries of the head—especially of the brain.

Liver Function Tests.—Fischler reviews the recent additions to the knowledge of the functions of the liver. He pleads for a routine examination of urine for urobilin, which reveals a concomitant disease of the liver as often as the test for albumin reveals disease in the kidney. Starvation as well as one-sided diets injure the liver. The empiric diet for gout—little protein and fat and moderation in general—is the best rest cure for the liver. Intravenous injections of glucose should not be forgotten in acute grave disturbances of the liver (acute atrophy, mushroom poisoning).

Wiener klinische Wochenschrift, Vienna

38: 1229-1252 (Nov. 12) 1925

Endogenous Dermatoses. G. Singer.—p. 1229.
Spirochetes in Vienna Rats. I. Takaki.—p. 1231.
*The Motor and Secretory Function of the Stomach. L. Hess and J. Faltitschek.—p. 1232.
Vasomotor Disease of the Internal Ear. H. Brunner.—p. 1235.
*Insulin and Cholin. K. Waltner.—p. 1237.
*Preparation of Vaccines. H. Popper and F. Silberstein.—p. 1237.
Specialists and Clinicians. E. Stransky.—p. 1241.
Surgical Mistakes of Practitioners. Schnitzler. Supplement.—pp. 1-24.

The Motor and Secretory Function of the Stomach.—Hess and Faltitschek found that patients with ulcers of the stomach show no change in the secretory or motor function of the stomach after paravertebral injection of 5-10 c.c. of a 1 per cent. solution of procain, made in the seventh thoracic segment. In healthy subjects and in gastric neuroses, the increase in both was constant. They believe that the lack of reaction in ulcer patients is due to an organic interruption of nerve conduction.

Insulin and Cholin.—Waltner found an increased susceptibility to cholin in animals during the insulin hypoglycemia.

Preparation of Vaccines.—Popper and Silberstein recommend quinin sulphate for preservation of vaccines.

38: 1253-1276 (Nov. 19) 1925

Ulcers of Stomach and Duodenum. G. B. Gruber.—p. 1253.
*Pepsin and Antipepsin in Ulcer and Nonulcer Cases. H. L. Popper.—p. 1259.
Protein and Sulphur Treatment. B. Dollinger.—p. 1260.
"Splints for the Humerus." C. Ewald.—p. 1264.
"Insulin and Leukocytes." A. Neumann.—p. 1265.
Color Photography in Medicine. E. Preissecker.—p. 1265.
Drug Treatment of Heart Insufficiency. E. Pick.—p. 1266.
Urgent Eye Surgery. V. Hanke. Supplement.—pp. 1-8.

Pepsin and Antipepsin in Ulcers and Nonulcer Cases.—Popper attributes the results of the Kohler antipepsin test only to the differing degrees of acidity of the gastric contents. The method has no diagnostic value.

Zentralblatt für Chirurgie, Leipzig

52: 2577-2640 (Nov. 14) 1925

*Peritonitis After Puncture of Common Bile Duct. A. Sohn.—p. 2578.
Strengthening of Vessel Wall. N. J. Gurewitsch.—p. 2581.
Lymphangioplastic Treatment of Elephantiasis. M. Sokolowski.—p. 2583.
Treatment of Hydrocephalus. M. Sokolowski and J. Irger.—p. 2586.
Treatment of Acute Osteomyelitis. A. Fischer.—p. 2589.
Ectopia of the Testis. F. Bernhard.—p. 2592.

Peritonitis After Puncture of Common Bile Duct.—For diagnostic purposes, Sohn punctured the choledochus with a fine needle, during a gastro-enterostomy operation. The patient died four days later from a purulent biliary peritonitis.

Časopis lékařů českých, Prague

64: 1609-1644 (Nov. 7) 1925

Partial Hydronephrosis. J. Rejsek.—p. 1609.
*Hemoclastic Crisis. G. Altschuller.—p. 1613.
Standardization of Tetanus Antiserum. Feierabend.—p. 1616. C'td.
*Tuberculosis of the Placenta. F. Horálek.—p. 1621.
Compression of Common Bile Duct. Kudrnáč.—p. 1624.
Physical Chemistry in Biology. V. Podroužek.—p. 1627.
Bacteriologic Vitamins. J. Stolz.—p. 1628.
Bismuth Treatment. A. Měska.—p. 1632.
Cleaning of Vaccine Virus. K. Urbánek.—p. 1635. C'cn.

Hemoclastic Crisis.—Altschuller considers the shifting of the leukocytes to the left (Arneth) as the most important sign of a true hemoclastic crisis in Widal's sense. He produced it by administration of sugar in diseases of the liver, as well as by Umber's method (milk by the rectum). No shifting was observed in the refractory phase following administration of milk, or after an enema consisting of water.

Tuberculosis of the Placenta.—Horálek found tuberculous nodes in the placenta of a woman who died two months after a normal delivery. The child was 5 months old at the time of the report, and developing normally.

Hospitalstidende, Copenhagen

68: 953-968 (Oct. 15) 1925

*Skin in Surgical Affections. J. Ipsen. Begun, p. 944.

Temperature of the Skin in Surgical Affections.—Ipsen describes his technic for recording the temperature of the skin under a layer of felt, and charts the findings under various conditions. Abnormally high temperatures at certain points, revealed by this technic, aid in differential diagnosis between loose bodies in joints or laceration of a semilunar cartilage and inflammatory processes, for example. The local temperature was exceptionally high with a sarcoma in the knee and with mammary cancers. The findings were not instructive in peritonitis.

Ugeskrift for Læger, Copenhagen

87: 881-904 (Oct. 8) 1925

*Transformation of Glycogen. Lundsgaard and Holbøll.—p. 881.
*Treatment of Genital Gangrene. O. Jersild.—p. 884.
*Schick Reaction in Greenland. E. Bay-Schmidt.—p. 889.
Analysis of Breads. H. Kærn.—p. 889.

Transformation of the Liver Glycogen Into Glucose.—Lundsgaard and Holbøll report research which shows that the glycogen in fresh and finely chopped liver tissue is split into alpha-beta glucose. Insulin does not affect this cleavage. They assume that in the living subject the glycogen undergoes the same transformation. The carbohydrates under the action of insulin are partly oxidized and partly deposited as glycogen. When the insulin is no longer in the blood, the deposits of glycogen are split into glucose, which explains the higher sugar content of the blood in the morning. Is the new glucose—ny glucose, as they call the kind with a lower specific rotating power—an intermediate link in the production and depositing of glycogen?

Neo-Arsphenamin in Treatment of Genital Gangrene.—Jersild gave the drug by the vein, in his twelve cases, and states that the effect on the temperature and the course of the process was so prompt and so constant that this treatment should be given at once in every case, regardless of the temperature or general symptoms.

The Schick Reaction in Eskimos.—Bay-Schmidt relates that a strongly positive reaction was obtained in an average of 68 per cent. of 382 persons in the isolated colony of Julianehaab. A dubious response was obtained in all the others, none giving a decidedly negative reaction. Diphtheria has appeared only once in the colony (1921) and soon died out, attacking only thirty young persons.

87: 905-922 (Oct. 15) 1925

*Action of Liver Tissue on Glucose. Lundsgaard and Holbøll.—p. 905.
Vaccine Therapy of Suppurative Colitis. A. Borgbjærg.—p. 909.
Case of Epiploitis Tumefaciens. E. Holm.—p. 912.
Fight Against Rural Tuberculosis. Kjer-Petersen.—p. 914.

Action of Liver Tissue and Insulin on Glucose in Vitro.—Lundsgaard and Holbøll conclude from their latest research that the liver serves in carbohydrate metabolism as a storage place for glucose in the form of glycogen, and has no direct importance for the metabolism of glucose in the organism. Contrary to muscle tissue, liver tissue is not able to form new glucose out of alpha-beta glucose under the influence of insulin.

JOURNALS ABSTRACTED IN THE CURRENT MEDICAL LITERATURE DEPARTMENT, JULY-DECEMBER 1925

Abstracts of important articles in the following journals have been made in the Current Literature Department of THE JOURNAL during the past six months. Any of the journals, except those starred, will be lent by THE JOURNAL to subscribers in the United States and to Fellows of the American Medical Association for a period not exceeding three days. Two journals may be borrowed at a time. No foreign journals are available prior to 1920, nor domestic journals prior to 1923. Requests for periodicals should be addressed to the Library of the American Medical Association and six cents in stamps should be enclosed. This covers the average expense of mailing a journal. Thus most of these journals are accessible to the general practitioner, no matter where located.

A. M. M., Asociacion Medica Mexicana: Continued as Revista de Asociacion Medica Mexicana.
Acta Chirurgica Scandinavica. Irreg. 20 kronor. Stockholm.
Acta Dermato-Venereologica. Irreg. $5.50 per vol. Copenhagen.
Acta Medica Scandinavica. Irreg. $5.50 per vol. Stockholm.
Acta Ophthalmologica. Irreg. $6 per vol. Copenhagen.
Acta Pathologica et Microbiologica Scandinavica. Irreg. $6. Copenhagen.
Acta Pædiatrica. Irreg. $5 per vol. Uppsala.
Acta Scholae Medicinalis Universitatis Imperialis in Kiota. Irreg. 3.30 yen per issue. Tokyo.
American Heart Journal. Bi-m. $7.50. St. Louis.
American Journal of Anatomy. Bi-m. $7.50 per vol. Philadelphia.
American Journal of Diseases of Children. M. $4. A. M. A., Chicago.
American Journal of Electrotherapeutics and Radiology. M. $5. Baltimore.
American Journal of Hygiene. Bi-m. $6. Baltimore.
American Journal of the Medical Sciences. M. $6. Philadelphia.
American Journal of Obstetrics and Gynecology. M. $6. St. Louis.
American Journal of Ophthalmology. M. $10. Chicago.
American Journal of Pathology. Bi-m. $5. Boston.
American Journal of Physiology. M. $5 per vol. Baltimore.
American Journal of Psychiatry. Q. $5 per vol. Baltimore.
American Journal of Public Health. M. $5. New York.
American Journal of Roentgenology and Radium Therapy. M. $10. New York.
American Journal of Syphilis. Q. $8. St. Louis.
American Journal of Tropical Medicine. Bi-m. $5 per vol. Baltimore.
American Review of Tuberculosis. M. $8 per vol. New York.
Anales de la Facultad de Medicina, Universidad de Lima. Bi-m. $1.50.
Anales de la Facultad de Medicina, Universidad de Montevideo. M. 6 pesos.
*Anales de Sociedad Médico-Quirúrgica del Guayas. M. $0.30 per issue. Guayaquil.
*Annaes Brasileiros de Dermatologia e Syphiliographia. Bi-m. 20 milreis. Rio de Janeiro.
Annales de l'Institut Pasteur. M. 60 francs. Paris.
Annales des Maladies Vénériennes. M. 60 francs. Paris.
Annales de Médecine. M. 60 francs. Paris.
*Annali di Neurologia. Bi-m. 40 francs. Naples.
Annals of Clinical Medicine. M. $7. Baltimore.
Annals of Medical History. Q. $8. New York.
Annals of Otology, Rhinology and Laryngology. Q. $6. St. Louis.
Annals of the "Pickett-Thomson" Research Laboratory. Irreg. Price varies. London.
Annals of Surgery. M. $10. Philadelphia.
Annals of Tropical Medicine and Parasitology. Irreg. £1 2s. 6d. per vol. Liverpool.
*Archiv Biologitcheskikh Nauk. Bi-m. 6 rubles. Moscow.
Archiv für Gynäkologie. Irreg. Price varies. Berlin.
Archiv für Kinderheilkunde. Irreg. 15 gold marks per vol. Stuttgart.
Archiv für Klinische Chirurgie. Irreg. Price varies. Berlin.
Archiv für Verdauungs-Krankheiten. Irreg. 18 marks per vol. Berlin.
Archives of Dermatology and Syphilology. M. $8. A. M. A., Chicago.
Archives Franco-Belges de Chirurgie. M. 60 francs. Brussels.
Archives de l'Institut Pasteur de Tunis. Q. Tunis.
Archives of Internal Medicine. M. $5. A. M. A., Chicago.
Archives des Maladies de l'Appareil Digestif et de la Nutrition. Monthly except August and September. 50 francs. Paris.
Archives des Maladies du Cœur, des Vaisseaux et du Sang. M. 50 francs. Paris.
Archives de Médecine des Enfants. M. 45 francs. Paris.
Archives Médicales Belges. M. 30 francs. Brussels.
Archives of Neurology and Psychiatry. M. $8. A. M. A., Chicago.
Archives of Ophthalmology. Bi-m. $6. New York.
Archives of Otolaryngology. M. $6. A. M. A., Chicago.
Archives of Surgery. M. $8. A. M. A., Chicago.
Archivio Italiano di Chirurgia. Irreg. 100 lire per vol. Bologna.
*Archivos Españoles de Enfermedades del Aparato Digestivo y de la Nutrición. M. 21 pesetas. Madrid.
Archivos Españoles de Pediatria. M. 25 pesetas. Madrid.
Archivos Latino-Americanos de Pediatria. M. $5. Buenos Aires.
*Ars Medica. M. 25 pesetas. Barcelona.
Atlantic Medical Journal. M. $3. Harrisburg.
Australian Journal of Experimental Biology and Medical Science. Q. £1 1s. Adelaide.
Beiträge zur klinischen Chirurgie. Irreg. Price varies. Berlin.
*Boletín del Instituto de Medicina Experimental para Estudio y Tratamiento del Cáncer. Bi-m. Buenos Aires.
*Boletin de Sociedad de Cirugia de Chile. Irreg. 20 pesos. Santiago.
Boston Medical and Surgical Journal. W. $6.
Brain, A Journal of Neurology. Irreg. 24s. London.
Brazil-Medico. W. $5. Rio de Janeiro.
Bristol Medico-Chirurgical Journal. Q. 10s. 6d.
British Journal of Anæsthesia. Q. $10. Manchester.
British Journal of Children's Diseases. Q. $6. London.
British Journal of Dermatology and Syphilis. M. 2 guineas. London.
British Journal of Experimental Pathology. Bi-m. £2. London.
British Journal of Medical Psychology. Q. $7 per vol. London.
British Journal of Ophthalmology. M. 2 guineas. London.
British Journal of Radiology. M. £2 2s. London.
British Journal of Surgery. Q. $10. Bristol.
British Journal of Tuberculosis. Q. $2.50. London.
British Medical Journal. W. 1s. 3d. per issue. London.
*Bruxelles Médical. W. 35 francs. Brussels.
Bulletin de l'Académie de Médecine. W. 50 francs. Paris.
Bulletin of the Johns Hopkins Hospital. M. $4. Baltimore.
Bulletin Médical. Semi-w. 25 francs. Paris.
*Bulletin of the Naval Medical Association. Q. Tokyo.
Bulletin of the Porto Rico Medical Association. Q. $3. San Juan.
Bulletins et Mémoires de la Société Médicale des Hôpitaux de Paris. W. 80 francs. Paris.
Calcutta Medical Journal. M. Rs. 5. Calcutta.
California and Western Medicine. M. $5. San Francisco.
Canadian Medical Association Journal. M. $6. Montreal.
Casopis Lekaru Ceskych. W. 170 č korun. Prague.
China Medical Journal. M. $10 Mex. Shanghai.
*Clinica. M. Montevideo.
*Clinica Medica Italiana. Bi-m. 42 lire. Milan.
*Clinica Pediatrica. M. 25 lire. Modena.
Colorado Medicine. M. $2.50. Denver.
Comptes Rendus des Séances de la Société de Biologie et de ses Filiales. W. 80 francs. Paris.
Crónica Médica. M. 6 pesos. Lima, Peru.
Dermatologische Zeitschrift. M. Price varies. Berlin.
Deutsche Medizinische Wochenschrift. W. 8.60 marks per quarter. Leipzig.
Deutsche Zeitschrift für Chirurgie. Irreg. $4.80 per vol. Leipzig.
Deutsches Archiv für Klinische Medizin. Irreg. $4.30 per vol. Leipzig.
*Echo Medical du Nord. W. 12 francs. Lille.
Edinburgh Medical Journal. M. 40s.
Encéphale. M. 80 francs. Paris.
Endocrinology. Bi-m. $6. Los Angeles.
Finska Läkaresällskapets Handlingar. Bi-m. 120 Finnish marks. Helsingfors.
Gaceta Médica de Caracas. Semi-m. 24 bolivares. Caracas, Venezuela.
*Gaceta Medica de Mexico. Irreg. 1 peso per issue. Mexico City.
Giornale Italiano de Dermatologia e Sifiliologia. Bi-m. 100 lire. Milan.
Glasgow Medical Journal. M. 30s.
*Grèce Medicale. M. 25 francs. Athens.
Gynécologie et Obstétrique. M. 70 francs. Paris.
Heart. Irreg. 37s. 6d. per vol. London.
Hospitalstidende. W. 30 kroner. Copenhagen.
Hygiea. Semi-m. 24 kroner. Stockholm.
Illinois Medical Journal. M. $3. Oak Park.
Indian Journal of Medical Research. Q. £1. 1s. 4d. Calcutta.
Indian Medical Gazette. M. Rs. 18. Calcutta.
Indian Medical Record. M. 7 Rs. 8 annas. Calcutta.
International Journal of Psycho-Analysis. Q. $6.50. London.
Irish Journal of Medical Science. M. £1 5s. Dublin.
Jahrbuch für Kinderheilkunde und Physische Erziehung. M. 18 marks. Berlin.
Japan Medical World (Nippon-No-Ikai Sha). M. $6. Tokyo.
Journal of Anatomy. Q. $10.75 per vol. London.
Journal of the Arkansas Medical Society. M. $3. Little Rock.
Journal of Bacteriology. Bi-m. $5 per vol. Baltimore.

W.—Weekly; Semi-w.—Semi-weekly; Tri-w.—Tri-weekly; M.—Monthly; Semi-m.—Semi-monthly; Bi-m.—Bi-monthly; Q.—Quarterly
*Cannot be loaned.

Journal of Biochemistry. Q. $5.50 per vol. Tokyo.
Journal of Biological Chemistry. M. $5 per vol. New York.
Journal of Bone and Joint Surgery. Q. $5. Boston.
Journal of Cancer. Q. 10s. 6d. Dublin.
Journal of Cancer Research. Q. New York.
Journal de Chirurgie. M. 90 francs. Paris.
Journal of Clinical Investigation. Bi-m. $5 per vol. Baltimore.
Journal of Comparative Neurology. Bi-m. $7.50 per vol. Philadelphia.
Journal of Comparative Psychology. Bi-m. $5 per vol. Baltimore.
Journal of Experimental Medicine. M. $10. New York.
Journal of the Florida Medical Association. M. $3. Jacksonville.
Journal of General Physiology. Bi-m. $5. New York.
Journal of Hygiene. Q. $10. London.
Journal of Immunology. Bi-m. $5 per vol. Baltimore.
Journal of the Indiana State Medical Association. M. $3. Fort Wayne.
Journal of Industrial Hygiene and Abstract of Literature. M. $6. Boston.
Journal of Infectious Diseases. M. $5. Chicago.
Journal of Iowa State Medical Society. M. $3. Des Moines.
Journal of Kansas Medical Society. M. $2. Topeka.
Journal of Laboratory and Clinical Medicine. M. $8.50. St. Louis.
Journal of Laryngology and Otology. M. 40s. Edinburgh.
Journal of Maine Medical Association. M. $2. Portland.
Journal de Médecine de Bordeaux du Sud-Ouest. Semi-m. 35 francs.
*Journal de Médecine de Lyon. Semi-m. 40 francs.
Journal of Medical Association of Georgia. M. $3. Atlanta.
Journal of Medical Society of New Jersey. M. $2. Orange.
Journal Mental Science. Q. 30s. London.
Journal of Metabolic Research. M. $10. Morristown.
Journal of Michigan State Medical Society. M. $5. Grand Rapids.
Journal of Missouri State Medical Association. M. $2. St. Louis.
Journal of Nervous and Mental Diseases. M. $10. New York.
Journal of Neurology and Psychopathology. Q. 30s. London.
Journal of Obstetrics and Gynæcology of the British Empire. Q. 44s. Manchester.
*Journal of Okayama Medical Society. M. Okayama.
Journal of Oklahoma State Medical Association. M. $4. Muskogee.
*Journal of Oriental Medicine. Q. Dairen, South Manchuria.
Journal of Parasitology. Q. $3. Urbana.
Journal of Pathology and Bacteriology. Q. 40s. Edinburgh.
Journal of Pharmacology and Experimental Therapeutics. M. $6. Baltimore.
Journal of the Philippine Islands Medical Association. M. $6. Manila.
Journal of Physiology. Irreg. Price varies. London.
Journal des Praticiens. W. 40 francs. Paris.
Journal de Radiologie et d'Électrologie. M. 75 francs. Paris.
Journal of Radiology. M. $5. Omaha.
Journal of South Carolina Medical Association. M. $3. Greenville.
Journal of State Medicine. M. 2s. per issue. London.
Journal of Tennessee State Medical Association. M. $2. Nashville.
Journal of Tropical Medicine and Hygiene. Semi-m. 30s. London.
Journal d'Urologie Médicale et Chirurgicale. M. 70 francs. Paris.
Journal of Urology. M. $4. Baltimore.
Kentucky Medical Journal. M. $5. Bowling Green.
*Kenya Medical Journal. M. 25s. Nairobi.
Kitasato Archives of Experimental Medicine. Irreg. 1.20 yen per issue. Tokyo.
Klinische Wochenschrift. W. 24.40 gold marks. Berlin.
Lancet. W. $12. London.
Laryngoscope. M. $6. St. Louis.
Lisboa Médica. M. 75 milreis. Lisbon.
Lyon Chirurgical. Bi-m. 60 francs. Paris.
Lyon Médical. W. 30 francs.
*Marseille-Medical. 3 issues per month. 25 francs.
Médecine. M. 20 francs. Paris.
Mededeelingen van Dienst der Volksgezondheid in Nederlandsch-Indië. Irreg. Batavia.
Medical Journal and Record. Semi-m. $6. New York.
Medical Journal of Australia. W. £2 5s. Sydney.
Medical Journal of South Africa. M. £1 5s. Johannesburg.
*Medicina. M. $5 Mex. Mexico City.
Medicine. Q. $5 per vol. net. Baltimore.
Medizinische Klinik. W. 36 gold marks. Berlin.
Memorias do Instituto Oswaldo Cruz. Irreg. 20 reis per issue. Rio de Janeiro.
Mental Hygiene. Q. $3. Albany.
Military Surgeon. M. $4. Washington.
Minnesota Medicine. M. $3. St. Paul.
Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie. Irreg. Jena.
Monatsschrift für Geburtshülfe und Gynäkologie. M. 18 marks per vol. Berlin.
Monatsschrift für Kinderheilkunde. M. Price varies. Leipzig.
Münchener Medizinische Wochenschrift. W. $8. Münich.
National Medical Journal of China. Bi-m. $2.50. Shanghai.
Nebraska State Medical Journal. M. $2.50. Lincoln.
Nederlandsch Maandschrift voor Geneeskunde. M. 16 florins. Leiden.
Nederlandsch Tijdschrift voor Geneeskunde. W. 29 florins. Haarlem.
Néoplasmes. Bi-m. 30 francs. Paris.
New Orleans Medical and Surgical Journal. M. $3.
New York State Journal of Medicine. M. $3.50. New York.
Norsk Magazin for Lægevidenskaben. M. 25 kroner. Oslo.
Northwest Medicine. M. $2. Seattle.
Nourrisson. Bi-m. 30 francs. Paris.
Occupational Therapy and Rehabilitation. Bi-m. $5. Baltimore.
Ohio State Medical Journal. M. $3. Columbus.
Paris Médical. W. 50 francs.
*Pathologica. Semi-m. $6. Genoa.
Pediatria. Semi-m. 80 lire. Naples.
*Pediatria-Archivio di Patologia e Clinica Pediatrica. Irreg. 160 lire. Naples.
Philippine Journal of Science. M. $5. Manila.
Policlinico (sez. chir.) and (sez. med.). M. 45 lire each; (sez. prat.). W. 90 lire. Rome.
Practitioner. M. 2 guineas. London.
Prensa Médica Argentina. 3 issues per month. $6. Buenos Aires.
Presse Médicale. Semi-w. 60 francs. Paris.
Progrès Médical. W. 70 francs. Paris.
Prophylaktitcheskaia Medicina. M. 10 rubles. Kharkov.
Public Health Journal. M. $2. Toronto.
Quarterly Journal of Experimental Physiology. 10s. per issue. London.
Quarterly Journal of Medicine. 35s. London.
Radiology. M. $5. St. Paul.
Repertorio de Medicina y Cirugía. M. $4. Bogotá, Colombia.
Revista de la Asociación Médica Argentina (including 5 special sections). M. 20 pesos. Buenos Aires.
*Revista de Asociacion Medica Mexicana. Bi-m. Mexico City.
*Revista de Beneficencia Pública. Q. 15 pesos. Santiago.
*Revista de Cirugia. Bi-m. 20 pesos. Buenos Aires.
Revista Española de Medicina y Cirugía. M. 26 pesetas. Barcelona.
*Revista Española de Obstetrica y Ginecologia. M. 22 pesetas. Madrid.
*Revista Española de Urología y Dermatología. M. Madrid.
*Revista Médica Latino-Americana. M. 6 pesos. Buenos Aires.
Revista Médica del Rosario. Bi-m. 10 pesos. Rosario, Argentina.
Revista Médica del Uruguay. M. 8 pesos. Montevideo.
*Revista Médica de Yucatán. Bi-m. $2. Merida.
*Revista de Medicina y Cirugia. M. 15 bolivares. Caracas.
Revista de Medicina y Cirugía de la Habana. Semi-m. $4.50. Havana.
*Revista Mexicana de Biología. Bi-m. $4. Mexico City.
*Revista Yucateca de Dermatología y Parasitología. Q. $2. Merida.
Revue de Chirurgie. M. 65 francs. Paris.
Revue Française d'Endocrinologie. Bi-m. 30 francs. Paris.
Revue Française de Gynécologie et d'Obstétrique. Semi-m. 50 francs. Paris.
Revue Française de Pediatrie. Bi-m. 50 francs. Paris.
Revue de Médecine. M. 60 francs. Paris.
Revue Médicale de la Suisse Romande. Semi-m. 22 francs. Lausanne.
*Revue Neurologique. M. 80 francs. Paris.
Rhode Island Medical Journal. M. $2. Providence.
Riforma Medica. W. 90 lire. Naples.
*Rinascenza Medica. Semi-m. 60 lire. Naples.
Rivista di Clinica Pediatrica. M. 60 lire. Florence.
*Russkaia Klinika. M. 16 rubles. Moscow.
*Salvador Medico. Bi-m. 1 colon per 4 issues. San Salvador.
Scalpel. W. 20 francs. Brussels.
Schweizer Archiv. für Neurologie und Psychiatrie. Irreg. 35 francs per vol. Zurich.
Schweizerische Medizinische Wochenschrift. W. 17.20 francs per half year. Basel.
*Sciencia Médica. M. 5 pesos. Rio de Janeiro.
Sei-I-Kwai Medical Journal. Bi-m. $2. Tokyo.
Semana Médica. W. $5. Buenos Aires.
*Seuchenbekämpfung. Bi-m. 6 Swiss francs. Vienna.
Siglo Médico. W. 25 pesetas. Madrid.
South African Medical Record. Semi-m. 31s. 6d. Cape Town.
Southern Medical Journal. M. $3. Birmingham.
Southwestern Medicine. M. $2. Phoenix.
Surgery, Gynecology and Obstetrics with International Abstract of Surgery. M. $12. Chicago.
Svenska Läkaresällskapets Handlingar. Q. 18 kroner. Stockholm.
Texas State Journal of Medicine. M. $2.50. Fort Worth.
Tijdschrift voor Vergelijkende Geneeskunde. Irreg. 8.50 florins. Leiden.
Tohoku Journal of Experimental Medicine. Bi-m. 8 yen per vol. Sendai.
Tubercle. M. 25s. London.
Ugeskrift for Læger. W. 30 kroner plus postage. Copenhagen.
United States Naval Medical Bulletin. M. $1.50. Washington.
United States Veterans' Bureau Medical Bulletin. M. Washington.
Upsala Läkareförenings Förhandlingar. Irreg. 10 kronor per vol.
*Vestnik Endocrinologii. Bi-m. 6 rubles. Moscow.
*Vestnik Microbiologii i Epidemiologii. Q. 6 rubles. Saratov.
*Vestnik Roentgenologii i Radiologii. Bi-m. 13 rubles. Leningrad.
Virginia Medical Monthly. M. $2. Richmond.
West Virginia Medical Journal. M. $3. Huntington.
Wiener Archiv für Innere Medizin. Irreg. Price varies. Berlin.
Wiener Klinische Wochenschrift. W. $4. Vienna.
Wisconsin Medical Journal. M. $3.50. Milwaukee.
Zeitschrift für Geburtshülfe und Gynäkologie. Irreg. Price varies Stuttgart.
Zeitschrift für Kinderheilkunde. Irreg. Price varies. Berlin.
Zeitschrift für Klinische Medizin. Irreg. Price varies. Berlin.
Zeitschrift für Krebsforschung. Irreg. Price varies. Berlin.
Zeitschrift für Tuberkulose. Irreg. 20 renten marks per vol. Leipzig.
Zeitschrift für Urologie. M. 24 marks. Leipzig.
Zeitschrift für Urologische Chirurgie. Irreg. Price varies. Berlin.
Zentralblatt für Chirurgie. W. 48 renten marks. Leipzig.
Zentralblatt für Gynäkologie. W. 48 renten marks. Leipzig.
Zentralblatt für Innere Medizin. W. 40 renten marks. Leipzig.

W.—Weekly; Semi-w.—Semi-weekly; Tri-w.—Tri-weekly; M.—Monthly; Semi-m.—Semi-monthly; Bi-m.—Bi-monthly; Q.—Quarterly
*Cannot be loaned.

SUBJECT INDEX

This is an index to all the reading matter in THE JOURNAL. In the Current Medical Literature Department only the articles which have been abstracted are indexed.

The letters used to explain in which department the matter indexed appears are as follows: "E," Editorial; "C," Correspondence; "MI," Medicolegal; "P," Propaganda for Reform; "ME," Medical Economics; "ab," abstract; the star (*) indicates an original article in THE JOURNAL.

This is a subject index and one should, therefore, look for the subject word, with the following exceptions: "Book Notices," "Deaths" and "Society Proceedings" are indexed under these titles at the end of the letters "B," "D" and "S." State board examinations are entered under the general heading State Board Reports, and not under the names of the individual states. Matter pertaining to the Association is indexed under "American Medical Association." The name of the author, in brackets, follows the subject entry.

For author index see page 2110.

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A.—Association
Acad.—Academy
Am.—American
Coll.—College
Conf.—Conference
Cong.—Congress
Conv.—Convention
Dist.—District
Hosp.—Hospital
Internat.—International
M.—Medical or Medicine
Nat.—National
Phar.—Pharmaceutical
Phys.—Physicians
Ry.—Railway
S.—Society
Surg.—Surgical or Surgeon, Surgery

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